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THE ROLE OF SURGERY IN THE TREATMENT OF PEPTIC ULCER*

BY ALBERT J. SULLIVAN, M.D.†

AN attempt is made in this article to define the gastroenterologist's views of the rôle surgery should play in the treatment of peptic ulcer. Since the beginning of the modern epoch of diagnosis and treatment of ulcer, with the work of Sir Berkeley Moynihan between 1901 and 1904, and especially since the development of an organized, efficient method of medical treatment by Sippy in 1911, there have been two separate and distinct schools of therapeutics. One has believed that an ulcer was a surgical problem only in dire emergency, the other that immediate surgery would save years of time usually spent on unsuccessful medical treatment and would achieve a cure in a relatively short time. In the past seven or eight years, however, these strict barriers have broken down and in most clinics of any size there is close coöperation between the internist and surgeon in the field of peptic ulcer. To their credit, let it be noted that the surgeons soon recognized that routine surgical treatment resulted in only 60 per cent. "good" results, 20 per cent. "fair" results and about 20 per cent. "failures", and rapidly called attention to the inadequacy of such treatment. Individual surgeons have felt that the choice of operation and the details of technique were responsible for these poor results and we have seen general swings from gastroenterostomy, to excision plus gastroenterostomy, then to pyloroplasty, later to pylorectomy and now to gastrectomy.

Coincident with these swings has been a gradual recognition by the surgeon that most cases are benefited if treated by the internist. Today we find most surgeons of note using surgery only when adequate medical care has failed. Balfour, Bevan, Horsley, Finney and Lahey are a few of those in this country who have repeatedly emphasized this foundation upon which the successful management of peptic ulcer must rest. Peptic ulcer is a chronic recurrent disease, fundamentally a medical problem, but frequently requiring surgical interference. For the purpose of illustration it can readily be compared to pulmonary tuberculosis, another chronic disease usually amenable to medical treatment but subject to recurrences after apparent "cures". Great strides in its treatment have been made recently, however, by applying surgical procedures to selected cases. Artificial pneumothorax, phrenicotomy and thoracoplasty are universally recognized adjuncts to the treatment of pulmonary tuberculosis, but no one advocates surgery as the sole method of treatment to the exclusion of medical care.

Most gastroenterologists are not surgeons, but at least 25 per cent. of the ulcer patients under their care have had, or are about to need, surgical interference. Of the medical specialties, few have such frequent need for major surgery as gastroenterology. Conversely, few major abdominal operations require such careful and prolonged postoperative medical care as do those for peptic ulcer. Because of the close relationships, we feel that the gastroenterologist should clarify and define his views on the rôle of surgery in the treatment of peptic ulcer in general, and the indications for surgery and the choice of operation in a particular case. No attempt will be made to inflict the internist's viewpoint upon the surgical profession, but rather to digest the prevailing but frequently divergent views of the surgeons themselves. Surgical textbooks and monographs, as a rule, give but one side of the picture, as each author tends to recommend one particular operation which, judged in the light of his personal experience and skill, gives the best results. Other surgeons, of equal ability but with less experience in fitting that particular operation to the individual case and less technical skill in its performance, discard it because of a high operative mortality and poor end results. It is only by study of late results from many clinics that a true perspective can be obtained.

First, we must consider those indications for surgical intervention which are universally recognized by both internists and surgeons.

Indications for Surgery in Gastric Ulcer

Perforation.
Hemorrhage, repeated or severe.
Possibility, after thorough investigation, of the lesion being malignant.
Hour-glass deformity.
Pyloric obstruction.
No healing or partial healing after adequate medical treatment.

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Recurrence in spite of adequate medical treatment.

Indications for Surgery in Duodenal Ulcer

Perforation.
Hemorrhage, repeated or severe.
Pyloric or duodenal obstruction.
No healing or partial healing after adequate medical treatment.
Recurrence in spite of adequate medical treatment.

One of the frequent claims by the advocates of surgical treatment is that surgery will "save time". This unsupported statement, that the period of disability following surgical treatment is much less than the tremendous amount of time supposedly lost during medical treatment, has become widely accepted. A desire to save time has become an unofficial indication for surgery.

We know of no evidence which supports this prevalent opinion. On the contrary, there is an excellent impartial report by Forsyth[1] which indicates that surgery is followed by longer periods of disability than medical treatment. He analyses the disability claims, cures, relapses and deaths occurring in 59 cases of duodenal ulcer insured in the Medical Sickness, Annuity and Life Insurance Society. Members, all physicians, were free to choose medical or surgical treatment for their ulcers. Twenty-six chose medical and 33 surgical treatment. The average period of invalidism per case was 5 months after surgical and 4½ months after medical treatment. Thirty-three per cent. of operative cases were invalided from work for more than 5 months, whereas only 12 per cent. of the medically treated cases were so disabled. The cases were followed from one to twelve years and there were 36 per cent. recurrences after surgery and 38 per cent. after medical treatment. However, 4 of the surgical cases required second operations and there were three deaths in the surgical group,—two immediately following operation. There were no deaths in the medically-treated group.

Perforation.—Acute perforation, whether of a duodenal or gastric ulcer, is an indication for immediate surgical intervention. Early diagnosis and early operation are of prime importance. Statistics on large numbers of patients with perforated ulcers show that, if operation is undertaken less than six hours after perforation, the mortality is less than 5 per cent. If delayed from six to twelve hours, it increases to about 15 per cent., and the mortality of those cases delayed more than 12 hours approximates 50 per cent.

There is some disagreement as to the extent of the operative procedures to be employed in acute perforations. Most surgeons feel that only simple closure should be carried out, as any further procedures add considerably to the mortality and are not warranted. It must be emphasized that closure of a perforation is a life-saving procedure, not a cure for ulcer, and it is to be expected that other forms of therapy, medical or surgical, will later be necessary. In certain cases where the patient is in very good condition, and has reached the operating table within a few hours of perforation, more radical operations may be undertaken with little added risk. It is also true that in perforated duodenal ulcers which show a considerable degree of obstruction of the pyloric or duodenal canal at operation, or which are likely to develop obstruction because of the infolding and suture of the perforation, the addition of a gastro-enterostomy is warranted unless the patient is an exceedingly poor risk. Prepyloric gastric ulcers, because of the danger of obstruction, must occasionally be treated in the same fashion. In the great majority of perforated gastric ulcers, however, obstruction is not a factor. Simple closure is sufficient and the addition of a gastro-enterostomy means a definite increase in mortality without any compensatory physiological or clinical advantage being gained. This will be brought out later in the discussion of the surgical treatment of corporic gastric ulcer.

Hemorrhage. — The mortality from hemorrhage treated by medical means alone is in the neighborhood of 2.5 per cent., according to Hurst and Stewart[2]. The mortality from surgical intervention during acute bleeding is at least 30 per cent. In an equal number the bleeding point cannot be located.

The almost universal opinion of surgeons is best expressed by the following quotations. Balfour[3] states that, "The danger of succumbing to hemorrhage is less than the danger of operation during hemorrhage." Lahey[4] believes that, "There is no situation which is more likely to be followed by fatality than immediate operation for bleeding gastric or duodenal ulcer."

Thus we have a well-established rule that surgical intervention is contraindicated while the patient is still bleeding. But, as with all rules, there are occasional exceptions. Given an elderly man with a bleeding ulcer of known location, who has a degree of arteriosclerosis likely to interfere with the contraction and plugging of the bleeding arteriole, in whom the severe bleeding continues in spite of several days of fasting, lavage and adequate medical treatment, operative intervention after several transfusions may be the only method of preventing a fatality.

Bevan[5] expresses the usual point of view when he says, "After a patient has recovered from a massive hemorrhage, a surgical cure of the ulcer is not only indicated but, as a rule, demanded." We would agree to this point of view if it could be shown, (1) that the ulcer, as the source of bleeding, can be excised in most instances; (2) when excision is not possible that

healing of the ulcer is more likely after surgical than after medical treatment; and, (3) that the risk of future hemorrhage is less after operative intervention than after adequate medical management. Such faith in the merits of surgery is wholly unwarranted. Balfour[6] has shown that in 1072 cases of duodenal ulcer in which operation was performed in the Mayo Clinic in 1921 and 1922, there were 184 (18 per cent.) with a history of proved gross hemorrhage. In only 22 per cent. of these was excision found possible. Balfour also reports hemorrhage occurring after operation in 13 per cent. of ulcers with a previous history of hemorrhage. Hohlbaum[7] reports that of cases in which gastroenterostomy was performed for duodenal ulcer, 20 per cent. were followed by severe and occasionally fatal hemorrhage, some immediately and others years later. Most of these were patients who had bled previous to operation. Emery and Monroe[8] state, in their analyses of 556 cases, "There were also more patients who bled following medical then following surgical treatment, but the difference in this regard is slight. Thus 12.9 per cent. bled following medical treatment and 8 per cent. following surgical intervention." The difference is indeed slight when one remembers that there was a 6.9 per cent. operative mortality, exclusive of perforations, in the 248 surgically treated cases. Hurst[2] discusses 26 cases of hematemesis or melena in 60 consecutive patients with gastric or duodenal ulcer admitted to the New Lodge Clinic. Of these 11, or 42 per cent., occurred after operation, and of these, 4 had not previously bled. He concludes, "These figures show that the liability to hemorrhage is just as great after operation has been performed for a gastric or duodenal ulcer as after medical treatment, whether the patient has already had a hemorrhage or not."

A patient who presents himself with a bleeding ulcer and gives a past history of hemorrhage is not a surgical problem until after thorough medical treatment has failed to produce results. If the patient, however, has been undergoing adequate medical treatment when the hemorrhage occurs, he is a candidate for surgery, but not for the usually ascribed reason, namely, that surgery is the best method of preventing future hemorrhage. Rather it is because hemorrhage under such conditions indicates activity in the ulcer and a failure of the medical management. Let us not advocate surgery for hemorrhage, *per se*, except in the case of an active ulcer which fails to respond to medical treatment, recommending of course, the actual excision of the ulcer, when possible, because of its hemorrhagic tendencies.

Pyloric and Duodenal Obstruction. — Duodenal obstruction usually occurs within an inch of the pylorus. For all practical purposes it can be considered the same as pyloric obstruction and is usually secondary to duodenal ulcer. In 63 cases of pyloric and duodenal obstruction due to ulcer in 4,000 consecutive autopsies at Leeds[2], 84 per cent. were secondary to duodenal and 16 per cent. to gastric ulcer.

It should be emphasized that only cases of obstruction due to cicatricial deformity should be subjected to operative interference. A large percentage of cases showing six hour residue of the roentgen test meal are active ulcers with spasm, edema and congestion as chief factors in the temporary obstructive picture. Most of these respond surprisingly well to medical therapy, with a complete disappearance of clinical and x-ray evidence of obstruction in a week or ten days. Clute[9] found pyloric obstruction in 6 per cent. of 500 ulcer cases at the Lahey Clinic. Half of these were due to spasm and edema and were relieved by medical treatment alone. Medical care should always be given a fair trial, for even though a given case turns out to be one of true cicatricial obstruction after several weeks' treatment, the patient can be turned over to the surgeon an infinitely better operative risk.

The surgical treatment of true pyloric obstruction by gastroenterostomy is the brightest spot in the therapy of ulcer. Follow-up studies after a period of years yield in the neighborhood of 90 per cent. good results, a figure rarely reached by thorough medical treatment of uncomplicated chronic ulcers. It was the success of this procedure which led to the wave of surgical treatment of all types of peptic ulcer that reached its height ten or fifteen years ago and from which we are only now recovering.

It is difficult to find in the literature end results interpreted in the light of preoperative findings. We have only unanimous opinions of the great value of gastroenterostomy in pyloric obstruction due to duodenal ulcer. In a review of 472 surgically treated cases of peptic ulcer followed by Fremont-Smith and McIver[10] we are able to find some confirmatory evidence. Of 261 cases of duodenal ulcer operated upon, 66 per cent. of the patients were entirely free from symptoms for the period of observation (2 to 10 years); 72 showed "considerable six hour retention of bismuth". In this latter group there were 80 per cent. cures. If these 72 cases are subtracted from the total of 261, we have 60 per cent. cures in the group without six-hour residue and 80 per cent. in the group with retention, a wide difference of 20 per cent. Were it possible to select from these 72 cases those which had true organic stenosis, in contradistinction to those whose retention was due only to spasm and edema, the difference might be even more striking.

In the relatively small number of cases in which the obstruction is due to gastric ulcer the surgical procedure employed is usually more radical than simple gastroenterostomy for two

reasons. First, gastroenterostomy for gastric ulcer is not as successful as for duodenal ulcer, perhaps because the ulcer is still exposed to unneutralized gastric juice. Secondly, cancer is most frequently found in the pyloric end of the stomach and a pylorectomy or partial gastrectomy will remove the vulnerable tissue with but a slight increase in the operative mortality.

Hour-glass Deformity of the Stomach.—In comparison with the complications of hemorrhage, perforation and pyloric obstruction, hour-glass constriction of the stomach is relatively infrequent. When it occurs, however, the obstruction may be almost complete and the complications of a very serious nature.

Hurst[2] gives an excellent summary of the indications for operation in hour-glass stomach as follows:

"(1) With active ulceration. An operation should be performed without delay if the x-rays show that four-hour stasis occurs in the proximal segment. Otherwise medical treatment may be tried in the hope that part of the narrowing is caused by the spasm.

"(2) With no active ulceration. An operation should be performed if sufficient stasis is present to cause symptoms in spite of care in diet.

"(3) Whether the ulcer is active or not, an operation is indicated if the hour-glass contraction is associated with pyloric obstruction."

Gastrogastrostomy is the operation of choice, unless there is coëxistent pyloric obstruction and a gastroenterostomy is also necessary. There are some who feel that gastrectomy is the best procedure, in spite of a higher operative mortality.

Carcinoma Arising from Gastric Ulcer.—Space does not permit us to analyze the evidence in the controversy as to the frequency with which cancer arises on the basis of a chronic gastric ulcer. The reader is referred to the excellent articles on this subject by Ewing[11], Cabot and Adie[12], and Stewart[2]. Suffice it to say that the huge majority of internists, surgeons, and pathologists believe that no more than 10 per cent. of chronic gastric ulcers become malignant and a considerable number feel that 5 per cent. is a truer estimate. Recent articles by Hurst[13] and Bloomfield[14] on the rôle of achylic gastrica in the etiology of carcinoma of the stomach have shed new light on this problem.

As a result of this point of view, we no longer see the medical profession advising gastrectomy for all cases of gastric ulcer because the lesion may have, or may develop, carcinomatous changes. Such routine excision would save no lives, for gastrectomy itself carries a mortality of at least 10 per cent., even in the hands of the most competent of gastrointestinal surgeons.

It is well recognized that statistics are of little value in the handling of an individual case. The usual clinical history, physical examination, gastric analysis, blood count and even Roentgen-ray examination will not place all cases into one of the two categories, ulcer or cancer. At least 15 per cent. are borderline cases and to the conservative clinician nearly half of the true ulcers may have features suggestive of carcinoma. Should all these be submitted to radical surgery? Cannot some preoperative differentiation be made?

More and more clinics are adopting the therapeutic test, the method by which these cases are handled at the Lahey Clinic and which is described by Jordan[15]. The summary of her article is as follows:

"Gastric ulcers need not all be regarded as malignant or potentially malignant.

"Frank ulcers which show a complete disappearance by Roentgen examination and complete clinical relief may be regarded as healed.

"A certain percentage of frank ulcers are of the callus and penetrating type, and although these may partially heal and clinical symptoms be completely relieved, they are still discernible in a careful Roentgen examination as minute defects, and should be resected because they are a source of chronic irritation in an organ susceptible to malignant degeneration, and because, in my opinion there will later be recurrence of symptoms and recrudescence of the ulcer crater.

"A certain group of gastric lesions can be diagnosed as suggestive of malignancy, and because of this suspicion, even if palpation and visualization with laparotomy leave the surgeon in doubt as to their malignancy, they should be submitted unhesitatingly to radical surgery after a two or three weeks' period of pre-surgical treatment during which there has been a failure of fulfilment in one or more of the following requirements:

1. *Occult blood must disappear completely from the stools.*
2. *Symptoms must be completely relieved.*
3. *The Roentgen defect must disappear completely, and the contour and tone of the stomach be restored to normal.*

"Surgery for gastric lesions, except for palliative gastroenterostomy for inoperable malignant growths, should always be radical resection, since all lesions that are not potentially malignant are medically healable.

"A certain number of lesions, at first considered suggestive of malignancy, will be proved benign, tractable ulcers and may be safely treated medically because they fulfill the requirements outlined."

Out of 133 cases of gastric lesion admitted to the Lahey Clinic, 66 were immediately diagnosed as frankly malignant growths. Of the remaining 67 cases, 44 were tractable ulcers and responded to medical treatment, and 7 were thought to be ulcers but did not respond and were operated upon. Pathological examination

in the 7 cases did not show malignancy. In one other case gastrogastrostomy was required for hour-glass constriction. The remaining 15 cases did not respond to the presurgical treatment and were considered malignant. Of these, 14 showed malignancy at operation and in one case in which pathological specimens were not available the patient died after a palliative gastroenterostomy.

MEDICAL FAILURES

So far we have discussed the most tangible indications for surgical intervention, the organic complications. Most internists and surgeons will find themselves in fair agreement with the principles laid down in the preceding paragraphs. It is true, however, that some very excellent surgeons, while agreeing in principle, on being confronted with a given case of hemorrhage will operate as soon as the patient is in good shape; will perform a gastroenterostomy for obstruction due to spasm and edema; or resect the stomach for a chronic ulcer before trying medical treatment, in order to "save time" which might be spent in trying medical care, a form of therapy in which they have little faith. Much of this lack of confidence in the other man's work is due to the fact that the surgeons see only medical failures and the internists get the surgical failures. It is reassuring, nevertheless, in the study of the literature to find that the surgeons who are most emphatic in desiring thorough medical care before resorting to surgery are those who are carefully studying their end results over long periods. In such clinics there is always a close coöperation between internist and surgeon which rapidly brings returns which redound to the credit of both.

About 10 per cent. of all peptic ulcers develop the complications discussed above. We now come to the class which is much more difficult to define and discuss, the cases which do not respond to thorough medical treatment and those which recur in spite of continued adequate medical therapy. What constitutes thorough medical therapy? What are the criteria, especially in duodenal ulcer, which indicate healing? What constitutes adequate therapy after healing takes place? Do a few days of symptoms following dietary indiscretions or an acute coryza constitute a recurrence? Does one moderately severe recurrent attack of ulcer symptoms after years have elapsed mean that medical treatment has failed? Until internists can satisfactorily answer all these questions, medical therapy is haphazard and surgeons are justified in their poor opinion of it and will continue to feel that all the cases they see are "medical failures".

It is not the purpose of this article to discuss medical treatment, but in dealing with the subject of medical failures certain of these questions must be answered briefly.

1. Thorough medical treatment for an active ulcer requires eight to ten weeks of carefully supervised dietary control, the essentials of which are frequent feedings of bland food, antispasmodics, and in some cases, alkali therapy. In the mild and moderately severe cases treatment may be ambulatory, if circumstances warrant.

2. Disappearance of all gastric symptoms, disappearance of occult blood from the stools, gain in weight, and in gastric ulcer complete disappearance of roentgen evidence of ulcer, are the criteria of healing.

3. At least three years on a six-meal bland diet, with return to more frequent feedings during times of mental or physical stress (overwork, common colds, etc.), care of the bowels, avoidance of excesses in tobacco, total abstinence from alcohol, and eradication of foci of infection, constitute adequate after-therapy.

4. A fairly liberal diet, avoiding an excess of roughage and considering individual idiosyncrasies but retaining the mid-morning, mid-afternoon and bedtime glass of milk, should be followed permanently after the third year.

5. A few days of mild symptoms after mental or physical stress, dietary indiscretion, a period of severe constipation or an acute infection do not constitute a recurrence. Persistence of these symptoms longer than a week, in spite of efforts at control, demand stool and Roentgen-ray studies.

6. No general rule can be promulgated to deal with all cases in which active ulceration returns three or more years after true healing. Circumstances of the recurrence, age of patient, location of ulcer, etc., demand careful analyses in each case.

Does definition of such terms as "adequate medical treatment" enable one to draw a magic circle around certain cases and to declare that all others should be treated surgically? Not if we are to be fair to both patients and surgeons. First, let it be said that it is exceedingly rare for an uncomplicated ulcer to persist unhealed in spite of adequate medical treatment. No more than 2 to 3 per cent. fail to heal. It is impossible to estimate the number which recur in spite of adequate treatment, because of a paucity of reported end results over a five to ten year period, correlated with the degree of medical treatment. It is well known that the majority of patients will discontinue the medical regimen within a year or so after all symptoms cease, unless carefully followed by their physicians.

A striking correlation between the length of treatment and end results is suggested in the study of Emery and Monroe[8] on 556 cases of peptic ulcer treated at the Peter Bent Brigham Hospital. Of 18 patients receiving the full Sippy regimen for four weeks in the hospital and who discontinued it on discharge, follow-

up showed only 42 per cent. "good and improved" results. Of 28 patients who followed the full Sippy treatment faithfully for six to twelve months, there were 78 per cent. "good and improved" results. It is conceivable that four weeks of treatment might be of considerable value in the handling of acute ulcers, but in the management of chronic ulcers of several years' duration it must be considered as "no treatment", practically speaking. In the same study Emery and Monroe include a group of 43 patients who, for various reasons, received no treatment whatsoever. This group, which serves as a control, actually showed 49 per cent. "good and improved" results, a slightly better figure than the group which received only four weeks of treatment.

Is 58 per cent. of this latter group, which received only four weeks of treatment, to be subjected to immediate surgery because medical treatment has failed? No; it is the medical profession which has failed, not the method of therapy. If these had been cases of pulmonary tuberculosis, the hospital staff, the family physician, the insurance company and various public health organizations would have combined to see that treatment was continued, but these 18 cases of a comparably chronic disease were presumably dropped after a few weeks of treatment. Is it any wonder that 58 per cent. received no permanent relief? The point to be emphasized is that such patients, on their next visit to the physician or clinic for relief, unless complications have developed in the interim, are still eligible for their *first* course of thorough and adequate medical treatment.

Before leaving the subject of medical failures, I should like to mention a type of patient which is seen all too frequently by internists. This group occurs not only in the dispensary class of patients, but also among those who are economically better off. The typical member has a chronic ulcer of five or ten years' duration, with recurrences once or twice a year. When symptoms are severe, he cheerfully submits to a fairly vigorous medical regimen; when symptoms disappear, he returns to his former diet, habits of eating, smoking and drinking, in spite of the admonishments of his medical adviser. Aided by soda, induced vomiting, or even the stomach tube for occasional attacks of nocturnal pain, he keeps reasonably comfortable for six months or a year until a severe attack again requires vigorous management. Some of these cases eventually develop complications, but it is our opinion that, except for such complications, surgery offers only a fair chance of improvement since enslavement to appetite and vicious habits prevent proper postoperative medical care.

SURGICAL TREATMENT FOR UNCOMPLICATED ULCER

We have previously stated that about 10 per cent. of all peptic ulcers develop complications requiring surgical treatment. Between 5 and 10 per cent. more require surgical intervention because of failure to heal, or recurrence after thorough medical treatment. The selection of operative procedure to be used in such cases is one of the most difficult problems confronting the abdominal surgeon today. Factors which influence the choice of operation are:

1. *Location of ulcer in stomach or duodenum.*
2. *Position and size of ulcer.*
3. *Physiological end results.*
4. *Mortality and operative procedure.*
5. *Postoperative complications; jejunal ulcers, fistulas, etc.*
6. *Experience of the surgeon.*
7. *Condition of the patient.*

There is a considerable disagreement among surgeons as to the relative merits of the individual procedures. We shall point out only the most pronounced faults and virtues of the more common procedures.

GASTRIC ULCER

Simple Excision. — Of all the surgical procedures for peptic ulcer this is the least effective. The removal of the relatively small ulcerated area does not affect the underlying conditions which brought about ulceration and a recurrence is almost inevitable. Emery and Monroe[8] report a case in which a recurrence occurred five days after excision in the exact spot, marked by a silver clip, in which the first ulcer was located. In fifteen cases of simple excision of gastric ulcer which they report there were only 27 per cent. satisfactory results. Fremont-Smith and McIver[10] report only 39 per cent. good results in thirty-three cases. Dobson[16] had four recurrences out of ten cases, and Collinson[17] fifteen out of thirty-nine. It is clear that a low operative mortality does not justify an operation with such poor end results. Moynihan[18] stated in 1926, "I have abandoned the operation (simple excision) altogether in cases of gastric ulcer. The disappointments it brings are too many, and are neither easy to foresee nor certainly to be prevented."

Gastroenterostomy. — This was the routine method of treating gastric ulcers for many years but is considered of little value now. In the absence of pyloric obstruction it is of little physiological value. Many experiments have shown that if the pylorus is patent, nearly all of the stomach contents leave by that route. It has been said that in these cases the gastric ulcer may heal, not because of the gastroenterostomy, but in spite of it. Hurst[2] has collected 20 cases from the literature and from personal experience, in which a gastric ulcer developed after a gastroenterostomy for duodenal ulcer, there being no ulcer present in the stomach at the first operation. He rightly concludes, "As it is thus possible for an ulcer to develop for the

first time in a stomach after gastroenterostomy, it is hardly logical to perform the same operation for the cure of one which is already present."

Moynihan[18] states, "In consequence of my experience I have abandoned gastroenterostomy alone in the treatment of chronic gastric ulcer."

Excision Plus Gastroenterostomy.—Two wrongs do not make a right, nor does a combination of two poor operations produce a good result. Excision plus gastroenterostomy is rarely employed by most surgeons. Sleeve resection, which is at best a magnified form of excision, has also been largely discarded.

Gastrectomy. — Again we quote from Sir Berkeley Moynihan[18], "After all is said and done, that operation for the treatment of chronic gastric ulcer will alone survive which with the smallest risk brings about a complete subsidence of all symptoms, a permanent freedom from any recurrence of the like disease, and immunity from any disorder associated with the operation in consequence of its mechanical or physiological effects. The operation which seems destined to fulfill all these indications is gastrectomy."

Balfour[19] has stated, "It is apparent that partial gastrectomy is becoming more and more the operation of choice in cases of chronic gastric ulcer."

Gastrectomy is rather an elastic term and has been used to cover everything from resection of the pyloric antrum to complete excision of the stomach. Since about 85 per cent. of the gastric ulcers occur on the lesser curvature in its middle third, gastrectomy usually requires excision of about two-thirds of the stomach. The chief disadvantage of the operation is its mortality. In the hands of excellent gastrointestinal surgeons this is between 5 and 10 per cent.[5]; when undertaken by good surgeons who employ it but occasionally, the mortality may reach 45 per cent.[20] Were one to apply the method routinely to all cases of gastric ulcer, the mortality of 10 per cent. or more would be prohibitive. But when it is reserved for the relatively small number in which all methods of medical treatment have failed, such a mortality is justified by the excellent results obtained in the surviving cases. Jejunal ulcers are a comparatively rare complication. Pernicious anemia is an exceedingly rare sequel, except after a total gastrectomy.

DUODENAL ULCER

Gastroenterostomy versus Duodenogastrectomy.—Surgical opinion on the treatment of uncomplicated duodenal ulcer is sharply divided. One group, represented by most American surgeons, minimizes the incidence of jejunal ulcer after gastroenterostomy, accepts its other faults and prefers it to duodenogastrectomy because of a low operative mortality. The other group, represented by most Continental surgeons and an American group centered around Mt. Sinai Hospital in New York, minimizes the mortality of the more radical operation and discards gastroenterostomy because of a tremendous incidence of gastrojejunal ulcers (18 to 34 per cent.)[21].

Dr. Thomas R. Brown[22] expresses the point of view of the first group in the following, "Frankly, I cannot see the justification of extensive gastric resection for a small duodenal ulcer. It seems to me that the punishment is out of proportion to the crime. The mortality must be large in the hands of many surgeons and I should think that going through life with the inevitable microgastria might entail quite as much dietetic restriction as a permanent nonsurgical ulcer cure."

Dr. Richard Lewisohn[23] sums up the experience of the second group as follows: "During the last five years, we have used the major operation of partial gastrectomy in practically every case of duodenal ulcer. We have been highly satisfied with our results, and would not turn back to gastroenterostomy as a routine procedure.—While we have had some failures and while some patients have not been entirely cured, we feel that in replacing gastroenterostomy by partial gastrectomy we have reduced the bad results following the surgical treatment of duodenal ulcers from 50 per cent. to about 5 per cent."

It is our belief that the true road lies somewhere between these paths. Mortality is an important factor. Gastroenterostomy carries about 2 per cent. mortality in the hands of an expert who performs hundreds of them in a year, but to the average good surgeon it is between 6 and 9 per cent. Gastrectomy in the hands of a few may be only 5 per cent.; in others' hands it rises as high as 10 to 45 per cent. The latter figures are, of course, prohibitive and explain most of the opposition to gastrectomy. Such a mortality will always prevent gastrectomy from becoming a routine procedure with the average surgeon. No surgeon is justified in performing an "extensive gastric resection for a small duodenal ulcer". The small duodenal ulcer very rarely fails to respond to adequate medical treatment and is not a candidate for surgery. Large, multiple chronic duodenal ulcers with frequent recurrences, in patients who are good medical risks, may be subjected to gastrectomy if the surgeon is especially qualified. Ulcer which recurs after one operation, or the development of a jejunal ulcer after gastroenterostomy, warrants gastrectomy.

Pyloroplasty.—Certain other operations are of considerable value in carefully selected cases. Pyloroplasty is one of these. An ulcer on the anterior surface of the duodenum and within one inch of the pylorus is frequently best handled by some form of pyloroplasty. Lahey[24] has recently stated, "The operation today, as yet un-

proved by time and by application in the hands of a variety of surgeons, but which to my mind offers the most logical hope for the future in the surgery of duodenal ulcer, is the pyloroplasty with large partial excision of the sphincter, as suggested by Horsley and by Judd. This procedure is at least sound in its principles, doing away as it does with the fundamental factor (the pylorus) in the persistence of ulcer, permitting of the discharge of gastric contents into the duodenum where they belong, and allowing alkaline duodenal contents to pass back into the stomach for neutralization of gastric acids. Its drawback is its lack of adaptability to all types of duodenal ulcers."

Gastroenterostomy Plus Division of Pylorus.—The success of gastroenterostomy in the presence of pyloric obstruction leads to an operative attempt to produce obstruction in all cases by division of the pylorus. While routine performance of this maneuver has not fulfilled expectations, it is still of some value in the field of duodenal ulcer.

POSTOPERATIVE MEDICAL TREATMENT

Let us remember that occasionally after surgery for gastric ulcer and usually after operative interference for duodenal ulcer, the patient still has his ulcer. Let us also remember that whether the ulcer was removed or not, the patient is still suffering from most of the conditions responsible for the ulcer; he still has his "ulcer diathesis". Medical treatment after operation is even more important than preoperative medical therapy. When medical treatment has failed, we call upon surgery to create new conditions favorable to the destruction of the "ulcer diathesis". Surgery does not replace medicine, but it is an indispensable adjunct to medicine in these cases. Practically every surgeon interested in the subject of peptic ulcer insists on postoperative medical treatment. Adequate postoperative medical treatment requires indefinite adherence to the régime (outlined above) required after successful medical therapy. Just as most so-called failures are due to inadequate medical treatment so are most surgical failures due to inadequate postoperative therapy. To return to our old comparison, do we expect pulmonary tuberculosis to be cured by any operative procedure unaided by medical care? Let us remember, then, that we can only expect a high percentage of cures of ulcer by long continued adequate medical treatment, reinforced by prompt and proper surgical interference when the indications arise.

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THE DIFFERENTIAL DIAGNOSIS OF SUBACUTE BACTERIAL HEART DISEASE AND BANTI'S DISEASE. CASE REPORT*

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THAT subacute bacterial heart disease (Streptococcus viridans) may simulate Banti's disease is evident from the following case history. A correct differentiation of the two diseases is important for if Banti's disease is present, splenectomy is indicated, and this operation is unwarranted in the Streptococcus viridans infection.

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CASE REPORT

The patient was a business man, aged forty-five years. His chief complaints were swelling of the abdomen, weakness, pain, insomnia, and afternoon fever. He was of superior intelligence and the history is deemed to be reliable.

Past history. At the age of twenty he was refused life insurance because of a "leakage of the heart".

He acquired gonorrhea when thirty years old but denies syphilis. He had tonsillitis followed by "sciatic rheumatism" at thirty-two years.

When a child he had measles, whooping cough, and scarlet fever. His history was negative for chorea, rheumatic fever and tonsillitis other than that mentioned above.

Present illness. A year ago he awoke, one morning, unable to read or speak. Complete recovery followed in the subsequent nine days. His physician told him that the disturbance was caused by a "plug in the vessels of the head or neck". He believes that his illness began insidiously somewhat more than a year ago.

Six months ago he had all his teeth extracted without apparent benefit of his run-down condition. He felt weak, was very nervous and couldn't sleep. There was a rise of temperature every afternoon to 100°-102.5° F.; the patient took his temperature at 4 P. M. and 7 P. M. daily. There have been occasional severe night sweats during the past six months.

Four weeks ago he noted gradual swelling of his abdomen and experienced pain for the first time during this illness. The pain was described as sharp and was especially prone to occur on coughing or turning in bed. It radiated from the right upper to the right lower quadrant. There were also vague pains along the left side of the abdomen and occasionally in the legs, where it was associated with small, discrete, red spots and swellings. He was told that these spots were due to purpura hemorrhagica.

He had had attacks of slight dyspnea. There were no digestive complaints; the stools were dark brown in color. There had been a loss of 37 lbs. in weight during the past year; the present weight was 131 lbs. He was nervous and worried, for which he believed he had adequate cause.

Physical examination. Very pale and cachectic-looking man. Visible pulsations of the carotid arteries. Mucous membranes pale; no jaundice.

Heart. Impulse somewhat heaving just outside left nipple line, 11 cm. from the midsternum; percussion of the left border of deep cardiac dulness corresponds. The right border of dulness in the fourth interspace is 4 cm. from the midsternum. There is a definite systolic thrill of moderate intensity over the cardiac impulse. A blowing systolic murmur is audible over the precordia and transmitted to the neck; a diastolic murmur, beginning with the second sound, is heard best along the left sternal margin and transmitted to the apex. Action regular, rate 86. Pulses collapsing in type; diastolic murmur present on pressure. Blood pressure 150/30.

Abdomen. Liver palpable, slightly tender, lower border three-fourths distance from ensiform cartilage to umbilicus. The spleen is easily palpable and extends to two fingers' breadth above the crest of the ilium.

There are a few petechiae on the lower legs. The remainder of the physical examination is normal.

Laboratory findings. Blood: white cells 3,270; differential count: polymorphonuclears, 72 per cent.; lymphocytes, 21 per cent.; endothelials, 6 per cent.; eosinophils, 0.5 per cent.; basophils, 0.5 per cent.; myelocytes, 0. Hemoglobin, 55-60 per cent. Red cells 2,330,000. Smear: poikilocytosis rare, anisocytosis very slight, anisochromia very slight, polychromia 0, erythroblasts 0. Clotting time, one minute; bleeding time, one and three-quarters minutes.

Wassermann reaction negative. Blood chemistry: Non-protein nitrogen 67 (one month later it was 33), urea nitrogen 30. Urine (six examinations): 1008-1013, albumen large trace once, very slight in others, sugar absent; red blood cells absent in three, 50-60 per high power field in one, moderate number in one, and numerous in one; casts—few hyaline in all, occasional epithelial in one. Blood culture negative at end of sixth day.

Electrocardiogram normal save for a definite thickening of the initial ventricular complex (the QRS).

The temperature varied from 99° in the morning to an average of 100°F. in the afternoon.

Subsequent history. A diagnosis of chronic rheumatic heart disease, with aortic insufficiency and probable mitral stenosis, was made. It was suggested that the present illness might be due to subacute bacterial heart disease engrafted upon the former and a blood culture was advised. This culture being sterile for six days, it was felt that one of two other suggested diagnoses—Banti's disease or splenic anemia—was probably correct. Blood transfusion and splenectomy were done as offering a possibility of saving the patient, who was obviously losing ground rapidly. Convalescence was reasonably normal for the first nine days. The neurologist then made the suggestion of encephalitic foci in the midbrain and medulla of infectious or hemorrhagic nature and usually transient. During the ninth to twelfth day there was paralysis of the left facial muscles, ptosis of the left eye, difficulty in speech and in swallowing. Death occurred on the twelfth day.

The heart action was satisfactory throughout and continued to beat three to four minutes after the respirations ceased.

*Autopsy.** (slightly abridged)

Pathological Diagnoses:

Heart: Coronary sclerosis and stenosis.
Healed infarcts.
Acute and chronic vegetative endocarditis.
Hypertrophy.
Brain: Cerebral hemorrhage (right),
Kidneys: Acute nephritis and healed infarcts.
Lungs: Hypostatic lobar and broncho-pneumonia.
Old healed pleuritis.
Old healed tuberculosis.
Liver: Hepatitis.
Gall Bladder: Chronic cholecystitis.
Acute general peritonitis
Spleen: Acute and Chronic Splenitis—Hematopoiesis.

6 cm. to the left of the umbilicus and extending between the costal margin and the symphysis pubis is a relatively recent surgical incision 19 cms. long partially closed by continuous catgut and interrupted silkwork suture. The gaping edges of this wound are gangrenous and covered with a thick purulent material.

Abdominal Cavity: Contains approximately 300 cc. of a slightly blood-tinged, rather thick, greenish, purulent material. Mesenteric lymph nodes are slightly enlarged and pale. Diaphragm on the right is at the 5th space; left 6th rib. Intestines are moderately distended with gas and fecal material and their serous surface, like all of the serous surfaces, slightly reddened and in places covered with soft, friable, easily removable "bread and butter" adhesions.

Pleural Cavities: Relatively negative except for a few old fibrous adhesions about the apices and base.

Pericardial Cavity: Contains no abnormal fluid or adhesions.

Heart: Weight 500 grams; measures 15 x 9 x 7 cms. Covered by a moderate amount of subepicardial fat. Coronaries stand out beneath the surface very markedly. The myocardium is firm, very dark reddish-brown in color. It is mottled in several places by irregular, translucent, grayish patches and by oc-

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casional round, white, tough fibrous patches, the largest of which measures 1 cm. in diameter. Endocardium is relatively negative except in the immediate region of the valves. Mitral valve is foreshortened and somewhat thickened. Covering its free margin are several soft, friable, shaggy, reddish-brown vegetations. Studding these vegetations are occasional small, round areas measuring approximately 1 mm. in diameter, the consistency of which resembles closely that of atheromatous material containing small granules of calcified material. There are larger areas, some measuring as much as 5 mm. in diameter, adjacent to the softer lesions on the valves and the chordae tendineae are roughened in many places by similar outcroppings. The cusps of the aortic valve are thickened and in places calcified. Also there are here lesions of a combined pultaceous and semi-calcified material. These are completely calcified at their base or point of attachment and are larger than those about the mitral valve, one of them measuring 1 cm. in length and about 5 mm. in diameter. The coronary arteries show advanced stages of atheromatous degeneration with many areas of calcification. In many places vessels are actually occluded by this process. Foramen ovule closed.

Valve Measurements

Tricuspid valve	12.4 cm.
Pulmonic valve	7.2 cm.
Mitral valve	9.0 cm.
Aortic valve	7.0 cm.
Left ventricular wall	2.0 cm.
Right ventricular wall	0.5 cm.

Lungs: Right, weighs 786 grams, measures 27 x 17 x 8 cms. The pleural surface is roughened at the apex and base by old fibrous adhesions. Aside from this the surface is smooth, glistening, and mottled various shades of dark and light red. Posteriorly all three lobes are dark red, non-crepitant and firm. Anteriorly they seem to be semi-crepitant, soft and boggy. Cut surface is almost uniformly dark red throughout. Alveoli are poorly defined and exude upon pressure much frothy and purulent bloody fluid. The bronchi are reddened and covered with a thin, watery, blood-tinged material. *Left*, weighs 735 grams; measures 26 x 15 x 6 cms.; similar in every respect to its fellow on the right.

Spleen: Removed at previous recent operation. Herewith is incorporated a copy of the surgical report.

Gross Description: Spleen weighs 428 grams; measures 16 x 11 x 5 cms. Firm, smooth, regular and dark reddish-brown. Cut surface pale reddish-brown. Germinal centers stand out prominently as translucent gray spots. Trabeculae well defined. Little pulp upon scraping. Surface has a distinctly meaty, firm appearance.

Microscopical Examination. Germinal centers throughout show much proliferated activity, occasional small islands of hyalinized material, some fibrin and infiltration of endothelials. Moderate increase throughout of the interstitial tissue. Many groups of plasma cells and definite evidence of hematopoiesis is outside of the germinal centers. Occasional vessel wall shows some fibrous and hyaline thickening.

Bacteriology. No growth in 72 hours.

Diagnosis:

Toxic Splenitis
Chronic Splenitis
Hematopoiesis.

Liver: Weighs 2120 grams, measures 31 x 21 x 1 cms. Distinctly enlarged and aside from where the surface is covered by soft, friable, easily removed, yellowish adhesions, is smooth, glistening and dark chocolate-brown in color. Cut surface is very tough, fibrous, dark chocolate-brown in color. Normal lobulations are not at all apparent.

Gall Bladder: Distended with approximately 75 cc. of a rather thin, clear, slightly bile-tinged mucous material. Wall is slightly thickened and apparently edematous. Mucosa is flecked with many minute points of bright yellow. No stones present; ducts patent.

Gastro-Intestinal Tract: Negative throughout its entire course, except for a marked engorgement of the vessels of the serosa and involvement of that membrane in a generalized acute abdominal process. There is a rather well defined autolysis throughout.

Pancreas: Weighs 90 grams; measures 16 x 5 x 2 cms. Not remarkable in gross.

Kidneys: Right weighs 215 grams; measures 13 x 6 x 3 cms. Rather soft, smooth, regular, dark reddish-brown. Cut surface: Pyramids poorly defined from the cortex, the whole being dark reddish brown. No differentiation can be made between the tubules and glomeruli. At a point near the upper pole the cortex is slightly indented to meet a wedge-shaped area of firm, yellowish white, fibrous tissue extending up from the pyramid with the base pointing outward. Capsule strips with ease leaving a slightly granular, somewhat pitted surface. *Left* weighs 735 grams; measures 26 x 15 x 6 cms.; similar in every respect to its fellow on the right except that it presents two areas of scarification of the cortex.

Adrenals: Not remarkable except for some postmortem cavitation.

Aorta: Shows a slight atheromatous degeneration throughout its entire course with a few small plaques about the arch.

Brain: Weighs 1560 grams. Scalp and calvarium are not remarkable. Dura strips with ease from the calvarium. Pia-arachnoids are markedly injected and these membranes are lifted from the surface of the cortex by an accumulation of a moderate amount of clear, serous, fluid. The gyri of the right hemisphere are flattened and the sulci are inconspicuous although they can be easily defined. This hemisphere is distinctly larger than the left and at a point about the junction of the frontal and temporal regions there is a distinct area of softening. Upon making multiple sections through the brain there is found a massive hemorrhage in the region of the right internal capsule, the internal striate artery in this region being completely destroyed by rhexis. Aside from this lesion and the surrounding softening, which involves practically all of the structures of this hemisphere, the remainder of the brain is negative. Actual blood clot at the region of the hemorrhage is 6 cm. in length and approximately 3 cm. in diameter.

Spinal cord: Negative.

Bacteriology: Peritoneal Cavity. B. Coli.
(Streptococcus viridans)

Heart's Blood: Streptococcus viridans.

Microscopical Examination

Heart: Slight fibrous thickening of the epicardium, beneath which are a few infiltrating lymphocytes, occasional plasma cells and endothelials. A similar infiltration seems to persist in the connective tissue about the large vessels as they dip into the myocardium. In spots the latter is completely replaced by somewhat edematous connective tissue. The endocardium shows a slightly vascular, marked fibrous thickening; in places this is hyalinized, while in others its surface is studded with small accumulations of fibrin, beneath which is a concentration of the cellular infiltration elsewhere generalized, consisting of lymphocytes, endothelials, occasional neutrophils and plasma cells. In the meshes of the fibrin are a few neutrophils and masses of organisms. The latter are Gram-positive cocci in short and long chains. Accompanying valve is similarly affected and infiltrated. It is vascular to its very tip.

Liver: The sinusoids about the central veins are moderately engorged. Numerous small places in which the liver cells are absent or are being invaded or removed by endothelials. Other areas in which the liver cords are pinched by an increase of connective tissue between the cells and the Kupffer cells. Serous surface is covered with a thin layer of organizing fibrin and infiltrated with large numbers of lymphocytes, plasma cells, and occasional endothelials.

Gall Bladder: Moderate engorgement of all vessels; occasional lymphatics plugged with neutrophils, some of which are infiltrating the surrounding tissue.

Kidney: Occasional small subcapsular areas in which the glomeruli are sclerosed, the tubules atrophic, and the interstitial tissue condensed and infiltrated with small numbers of lymphocytes and plasma cells. The remainder of the glomeruli are in various stages of degeneration. Some are completely sclerosed; some show irregular patches of sclerosis in the capillary tuft; some have dense adhesions between the tuft and capsule; some show a marked proliferation of the connective tissue of the capsule with a few remaining epithelial cells scattered between; many others show more acute disturbances such as thrombosis of the capillaries of the tuft, infiltration with neutrophils. Tubules are relatively negative; vessels throughout are markedly engorged. Occasional tubules in the pyramids contain casts of blood cells.

Adrenal: Essentially negative.

Aorta: Negative.

Bone Marrow: Moderate general hyperplasia. Erythroblastic derivatives predominate the picture.

Cerebrum: Most of the cortical material is degenerated and massively infiltrated with endothelial leukocytes. Most of them are phagocytic, while some others seem to be forming new capillaries. This cellular infiltration is immediately about a mass of clotted red cells.

Microscopical Diagnosis:

Heart: Fibrous myocarditis.
Chronic pericarditis.
Acute endocarditis.
Coronary sclerosis.
Lung: Broncho-pneumonia.
Healed pleuritis.
Pancreas: Chronic pancreatitis.
Liver: Chronic passive congestion.
Focal necrosis.
Gall Bladder: Early acute cholecystitis.
Kidney: Chronic vascular nephritis.
Acute glomerular nephritis.
Lymph Node: Acute lymphadenitis.
Cerebrum: Hemorrhage.
Bone Marrow: Erythroblastic hyperplasia.

DIFFERENTIAL DIAGNOSIS

A recent medical textbook[1] makes no mention of subacute bacterial heart disease in its presentation of the differential diagnosis of Banti's disease. Stevens[2], however, states that chronic infective endocarditis with multiple splenic infarctions may readily be mistaken for Banti's disease if fever, petechiae, and cutaneous erythematous nodes are absent, and leucopenia is present instead of leucocytosis. Certain of the findings will be discussed from the viewpoint of differential diagnosis.

Blood. In Banti's disease, according to Stevens[2], the anemia becomes more pronounced as the disease progresses, although the splenic enlargement sometimes precedes it by several years. The anemia is of the secondary type, with a low color index and usually leucopenia (3000-5000). The platelets are sometimes reduced and frequently there are found changes in the erythrocytes, especially anisocytosis, poikilocytosis, and normoblasts.

In subacute bacterial heart disease secondary anemia is usually present; sometimes this is severe enough to resemble anemia of the primary type. The white blood count varies; it may be 20,000 or considerably higher, or as low as 6,000 or less. It is not uncommon to find a difference of many thousand in the white count within a few hours. Occasionally there are found the large phagocytes, first described by Van Nüys[3]. These are large mononuclear cells, varying in size from 10 to 81 microns. The presence of these large phagocytes is practically certain evidence of subacute bacterial heart disease; the finding of one or two large mononuclear cells[4] (lymphocytes indistinguishable from those found in normal blood) with an ingested red cell is strong evidence of infection with the streptococcus viridans; and an increase in the number of large mononuclear cells is very suggestive of the disease. The platelets are abundant, and there is rarely any change in the coagulation or bleeding time.

Fever. The temperature is usually normal in Banti's disease, but slight fever may occur from time to time. Fever of the septic type is common in subacute bacterial heart disease; at times, however, there may be long afebrile periods.

Skin. Petechiae occur in about half[5] of the cases of the streptococcus viridans infection; their occurrence in Banti's disease is not mentioned by Stevens, who mentions merely the occasional occurrence of a brownish or grayish pigmentation of the skin. These petechiae may be found in the skin, the conjunctivae, the fundi of the eyes, and in the mucous membranes. Larger hemorrhages may occur. "Splinter hemorrhages"[6] under the nails are not common. Osler's nodes are fairly common; the Janeway spots are rare and more prone to occur in the acute form of bacterial heart disease. All these skin signs are inconstant and occur in crops.

Clubbing of the fingers and toes occurs in 70 per cent. of the cases of subacute bacterial heart disease; it may develop rapidly during the course of the disease.

Spleen. This organ is palpable in about seventy-five per cent.[7] of the cases of subacute bacterial heart disease. It is usually only moderately enlarged and sometimes detected only by means of skillful palpation. Occasionally it may be markedly enlarged, as in the above reported case, in which it extended well below the costal margin and was found to weigh 428 grams. In Banti's disease, which is often termed splenomegaly with anemia, the spleen is grossly enlarged and usually readily palpable.

Ascites. We are not aware that ascites oc-

curs in subacute bacterial heart disease. The fluid found in our case may have been the result of the abdominal operation.

Hemorrhages. Bleeding, usually from esophageal hemorrhages, occurs in about 50 per cent. of the cases of Banti's disease. It is not a feature of subacute bacterial heart disease.

Chills and Sweats. These are common in the Streptococcus viridans infection, but are not a feature of Banti's disease.

Heart. In subacute bacterial heart disease there are often murmurs and other signs of a previous infection,—usually that of rheumatic heart disease. The viridans infection is engrafted upon this and may cause additional murmurs. A rapid change in the cardiac murmurs is often observed. In some cases, however, cardiac murmurs are inconspicuous or absent.

In Banti's disease hemic murmurs may be present when the anemia is sufficiently advanced. In the case reported above there was sufficient anemia to have caused hemic murmurs.

Duration. A duration of ten years is not uncommon in Banti's disease, whereas subacute bacterial heart disease runs its course in a few months to about two years. Periodic remissions and exacerbations may occur in both.

Hepatic cirrhosis. This is a feature of the late stage of Banti's disease, whereas it is not in subacute bacterial heart disease.

Blood culture. The streptococcus viridans may often be found in the blood of patients with subacute bacterial heart disease. It needs emphasis, however, that the culture often must be incubated from four to seven days before the organism can be detected. The blood culture is sterile in Banti's disease.

DISCUSSION OF AUTOPSY FINDINGS*

After reading the protocol, it would seem that the case resolves itself very clearly into one having as its essential features a well advanced arteriosclerosis and a subacute bacterial endocarditis, with their respective sequelae. What clinical aspects of the case may have simulated Banti's disease is discussed elsewhere.

The heart presents lesions referable to both diseases, which must have given well-defined clinical evidence of their presence. The coronaries are sclerosed, in places to total occlusion, with resulting areas of extensive myocardial fibrosis, some of which encroach upon the conduction bundles. The endocardium is damaged by a bacterial process of long-standing, one that has unquestionably run the usual course of ups and downs of the viridans infections. The extent of time over which this has existed is further suggested by the areas of calcification and the soft, pultaceous vegetations. The mitral valve is shortened and shows a well-defined fibrous thickening, and the aortic cusps are involved by a moderate atheromatous and sclerotic process as well as the vegetative process described in the protocol. We again note that cultures from the vegetations showed the presence of streptococcus viridans and that the same organisms were there in profusion microscopically.

The infectious picture is sustained by the culture of streptococcus viridans recovered from the blood, the glomerular nephritis, the focal necrosis of the liver, the acute exacerbation of a chronic cholecystitis, the acute general peritonitis, the acute lymphadenitis, the splenic picture and the terminal bronchopneumonia. The hematopoiesis and active bone marrow also suggest an infectious picture of a subacute nature with a moderate secondary anemia.

The sclerotic syndrome reaches its climax in the terminal fatal lesion, a rupture of the lenticulostriate artery in the right hemisphere. There is an extensive sclerosis of all the cerebral vessels. This is all further accentuated by a well-marked arteriosclerotic condition of the kidneys, the aorta, and the larger vessels in many of the other organs.

The spleen is typical of the type usually found in low-grade infectious lesions. The one thing it lacks to complete the picture is any evidence of infarction. The organ is not excessively enlarged, weighing but 428 grams, is very firm, and its cut surface remarkable only for the prominence of the germinal centers. Microscopically the germinal centers are infiltrated with large numbers of endothelials, many of which are phagocyting, coarsely granular, golden-brown pigment. There is a general endothelial hyperplasia throughout and occasional small areas suggesting hematopoiesis. The wall of occasional arterioles is infiltrated with neutrophils and in the adventitia and surrounding tissue are small areas of hemorrhage, the cells of which are in all stages of degeneration. The sinusoids, aside from the endothelial hyperplasia, are not contracted, nor is the surrounding connective tissue increased. The pulp is infiltrated with many plasma cells and some lymphocytes. There is no thrombosis of the splenic vein, and the arterioles of the pulp show less evidence of sclerosis than most of the vessels elsewhere in the body. Entirely ignoring the rest of the clinical picture and considering the spleen alone, there is nothing here that suggests Banti's disease. There is a complete lack of necessary "fibroadeny". There is a great deal of pigment here, which some authors claim is never present and which in this instance can be traced directly to infectious vascular lesions. The spleen is relatively small, the majority of Banti's spleens weighing 1000 gms. or more, and the predominance of the infectious picture overrules all else.

*By C. F. Branch, M.D.

COMMENT

In retrospect of the case herein reported, it would appear that the original diagnosis of subacute bacterial heart disease should not have been so readily abandoned for that of Banti's disease. Too much attention was given to the large spleen and anemia, and too little to the petechiae, chills, details of the blood, fever, history of probable embolism, etc. Search should have been made for the large phagocytes and other details of the blood findings of subacute bacterial heart disease. The ocular fundi should have been examined.

More blood cultures would have been taken but for the fact that it was felt that the splenectomy should not be delayed if it was to be of benefit in this supposed case of splenic anemia. The spleen was unusually large for that of subacute bacterial heart disease, but similar cases have been reported. It is worthy of note that splenectomy has been suggested[8] for the therapy of subacute bacterial heart disease and actually performed[9] in one case, but without benefit. Our case records another instance of splenectomy without benefit.

The occurrence of hemorrhage of the brain in subacute bacterial heart disease is unusual, but a similar case has been reported[10].

It is also believed that this case warrants report to emphasize the similarity of Banti's disease and subacute bacterial heart disease, and the occurrence of the diagnostic error in spite of the fact that the correct diagnosis was originally entertained.

SUMMARY

A case is reported of subacute bacterial heart disease in which the diagnosis of Banti's disease was made erroneously.

Splenectomy was performed without benefit. Death resulted from cerebral hemorrhage.

The differential diagnosis, both clinical and pathological, is discussed.

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THE DIAGNOSIS OF PREGNANCY BY THE ASCHHEIM-ZONDEK TEST*

BY HENRY S. FINKEL, M.D.†

INTRODUCTION

THE clinical diagnosis of pregnancy during the early weeks frequently offers considerable difficulty. Early symptoms, such as absence of menstruation, morning nausea, fullness of the breasts and frequency of urination, are often due to other causes, while uterine changes during the first weeks are usually too slight to be recognized by bimanual palpation, even under the most favorable circumstances. With complicating factors, such as irregular bleeding, tumors, obesity and vaginismus, accurate diagnosis and proper treatment may be rendered even more difficult. The treatment of uterine bleeding due to threatened miscarriage, for example, differs radically from the treatment of the same condition due to tumors or inflammatory disease. Similarly extrauterine pregnancy and pelvic inflammation may present difficulties as to the proper choice of treatment. In many instances early diagnosis may not be essential from a strictly medical viewpoint, but may be of great importance in relieving the patient's mind.

Many attempts have been made to supplement the shortcomings of clinical diagnosis by laboratory tests, but none has thus far proved reliable, with the exception of the method of Aschheim and Zondek. In view of its high degree of accuracy, and its availability in the earliest weeks of pregnancy, I believe that the attention of the profession should be brought to this test, in order that it may be more generally utilized in suitable cases.

THEORETICAL FOUNDATION

In 1927, Aschheim and Zondek[1], investigating the stimulus that causes the onset of puberty, implanted various glands, tissues and fluids intramuscularly in infantile female white mice. All the implanted substances proved ineffectual, with the exception of the anterior pituitary gland. This tissue, four days after implantation, brought about all the changes characteristic of puberty; namely, opening of the vaginal orifice, cornification of the vagina, "heat", growth of the uterus, ovulation, and corpus luteum formation. The implantation of anterior pituitary gland into ovariectomized mice was without effect, proving that the action of the anterior pituitary gland was on the ovaries only, the uterine and vaginal changes being secondary to

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the liberation of ovarian hormone by the activated ovaries. They described the ovarian changes under three categories:

Anterior Pituitary Reaction I

Maturation of Graafian follicles and ovulation.

Anterior Pituitary Reaction II

Hemorrhages into Graafian follicles, discernible macroscopically as small hemorrhagic cysts, or "Blutpunkte." (Fig. 4.)

Anterior Pituitary Reaction III

The formation of corpora lutea, or corpora lutea atretica; i.e., luteinization of follicles with the ovum enclosed. (Fig. 2.)

From these experiments Aschheim and Zondek concluded that the internal secretions of the anterior pituitary gland furnish the stimulus to the periodic activity of the ovaries during the estrous cycle, and termed the anterior pituitary gland the "motor" of the ovary.

Since the anterior pituitary hypertrophies during pregnancy, they surmised that its secretion might be present in increased amounts during this period, a conjecture which was corroborated by the discovery that the urine and blood serum of pregnant women, injected in small amounts into infantile female white mice, produced changes identical with those caused by the anterior pituitary gland. The consistency with which the urine of pregnant women gave this reaction, and its absence in all other conditions, led them to utilize this fact as a diagnostic test for pregnancy. They found that the urine gave a positive reaction as early as five days after a skipped menstruation, and remained positive throughout pregnancy, with the exception of the last month, when the reaction was occasionally negative. A few days after delivery, or after the death of the embryo in utero, the reaction also became negative. Later they distinguished two components of the anterior pituitary hormone, calling them Prolan A and Prolan B. Prolan A is the hormone which causes follicle maturation (Anterior Pituitary Reaction I). Prolan B is the hormone which causes the formation of corpora lutea and "Blutpunkte" (Anterior Pituitary Reactions II and III). The "B" hormone is found in the urine only in pregnancy. The "A" hormone may be found in the urine in other conditions, such as menopause, amenorrhoea, endocrinopathy, genital tumors (benign and malignant), and pelvic inflammation. The presence of Prolan "A" is not indicative of pregnancy, therefore, but it has certain diagnostic significance.

It is interesting to note that the test is also positive in cases of hydatidiform mole and chorio-epithelioma, conditions in which there is chorionic tissue without an ovum. This leads to the supposition that the hormone is probably produced by chorionic tissue, an opinion which is supported by the fact that placental tissue gives a strongly positive reaction. The negative reaction after intrauterine death of the embryo may be explained by the concomitant cessation of placental function.

TECHNIQUE—The first voided urine of the morning is used for the test, since it contains the greatest concentration of hormone. The specimen, if not already acid, is slightly acidified with acetic acid and filtered. Five infantile female white mice, weighing between six and eight grams, are used for the test. Smaller mice should not be used because they are too immature to survive; larger mice should not be used because they may attain spontaneous ovulation during the test. The urine is injected into the mice subcutaneously, twice the first day, three times the second day, and once the third day, in doses respectively of .2, .25, .3, and .4 cubic centimeters at each injection. Not less than five mice should be used for each test, because one or more mice may die during the experiment from the toxicity of the urine, and one or more mice may fail to react in a positive case.

FIGURE 1. Ovary of control infantile white mouse, showing Graafian follicles in early stage of development.

Recently Zondek[2] has advocated the following modification:—Thirty cubic centimeters of acidified filtered urine are shaken vigorously for five minutes with ninety cubic centimeters of ether. The urine is separated off and allowed to stand for one hour in a wide vessel in order to allow all traces of residual ether to evaporate. The urine is then injected as above, except that graded doses are unnecessary and all the animals may be given .3 or .4 cc. The ether removes toxic substances from the urine, thereby reducing the mortality rate of the mice, and consequently increasing the accuracy of the test.

On the fifth day, 100 hours after the first injection, the mice are killed with illuminating gas, and the genital organs are inspected. In a negative case the genitalia present the normal

appearance for an infantile mouse. The ovaries, about pin-head in size, have a smooth grayish surface, the Y-shaped uterus is thread-like in appearance; the vaginal orifice is closed.

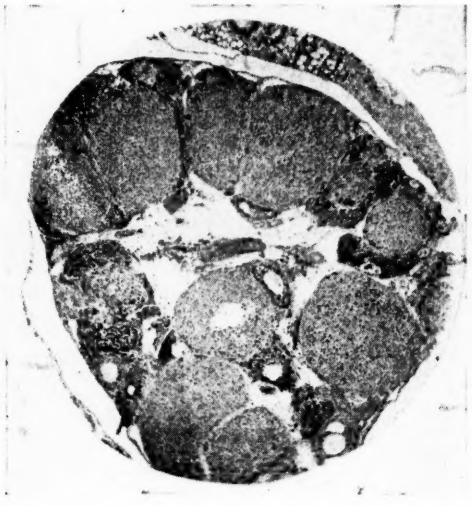

FIGURE 2. Ovary of infantile white mouse injected with pregnancy urine, showing numerous corpora lutea.

(Fig. 3b.) In a typical positive case, the following picture is seen: the ovaries are enlarged, hyperaemic, and studded with numerous pin-

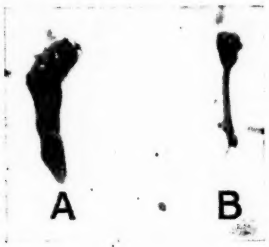

FIGURE 3. Ovary and uterine cornu of injected mouse (a) and control mouse (b). Note the enlargement of the uterus and ovary of the injected mouse, and the corpora lutea and corpora hemorrhagica studding the surface of the ovary.

point yellowish elevations (corpora lutea) and one or two brownish-red elevated blood points (hemorrhagic follicles); the uterine cornua are markedly distended and hyperaemic; the vaginal orifice is patulous. (Fig. 3a.) Occasionally the uterine and vaginal changes may be present without the ovarian changes. This condition is brought about by the presence of ovarian hormone in the injected urine, acting directly upon the uterus and vagina of the mouse. It is not diagnostic of pregnancy, however, since ovarian hormone is frequently present in varying amounts in the urine of women with normal menstrual cycles or even with amenorrhoea. If the preliminary washing of the urine with ether is done, any ovarian hormone that may be present is extracted by the ether, and this exogenous stimulation of the uterus and vagina does not occur. If there is any doubt as to the diagnosis from gross examination, the ovaries are sectioned serially and examined for the above changes. The presence of a single follicle showing luteinization or hemorrhage is pathognomonic of pregnancy. For practical purposes, it is rarely necessary to resort to microscopic examination.

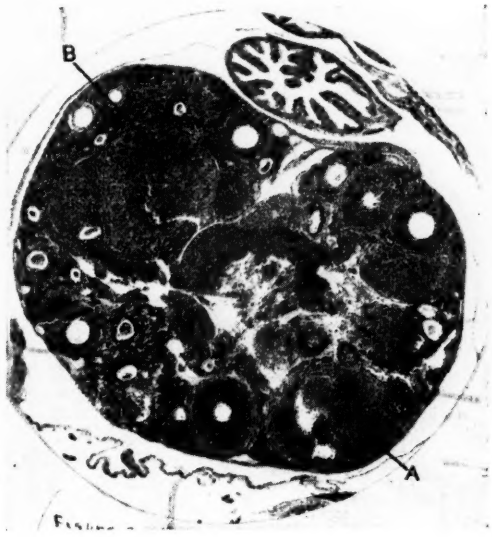

FIGURE 4. Ovary of infantile mouse injected with pregnancy urine × 150. Two follicles, partially luteinized, show extensive hemorrhage (a and b). Anterior Pituitary Reactions ii and iii. Positive pregnancy test.

RESULTS: I have applied this test in 64 cases, in most of which the specimens were submitted by physicians because the diagnosis was clinically uncertain. This report is based on those cases, 52 in number, in which the laboratory diagnosis was checked by subsequent clinical developments. Of the 21 cases of pregnancy the test was positive in 20 cases. The one negative result was obtained in a case in which the ovum had been dead for a few months preceding the test, a condition in which, as previously stated, the test is expected to be negative. This result, therefore, cannot be regarded as an error, but it must be recognized as a limitation of the test, since it fails to give a true insight into the clinical condition. From another point of view this limitation has its advantages; in cases of known pregnancy, when there is a question of death of the foetus, a negative test is strongly corroborative of this diagnosis. In eight cases a positive diagnosis was correctly made within five to six weeks from the last menstruation. In thirty-one cases of non-pregnancy the test was negative in every instance. The test was absolutely accurate, therefore in fifty-one out of fifty-two cases, or slightly more than 98%. These results conform closely with those obtained by Aschheim[3], who reports 98.8% accuracy in one thousand personal cases, and 98.3% accuracy in 1829 cases accumulated from the literature.

The mortality of the mice injected with the filtered urine was 6%, considerably less than

TABLE OF CASES

Case No.	Age	Past Menstrual Cycle in Weeks	Interval from Last Menstruation to Test in Weeks	Other Data in History	Physical Examination	Aschheim-Zondek Test	Subsequent Clinical Course
1	37	4	6	Married many years; sterile; no symptoms of pregnancy.	Not done.	Negative	Menstruated a few days after the test.
2	27	4-60	12	Frequent irregular bleeding since the last period, particularly following examination.	Inconclusive.	Positive	Pregnant
3	26	4	5½	No symptoms.	Uterus slightly enlarged; no softening.	Positive	Pregnant
4	22	4	2	Period lasted six days but much scantier than normal; fullness of breasts.	Uterus slightly enlarged and softened.	Positive	Pregnant
5	32	Lactation Amenorrhoea		Nausea.	Uterus slightly enlarged and very suggestive of pregnancy.	Negative	Not pregnant
6	30	4	6	Backache.	Fundus slightly enlarged.	Positive	Pregnant
7	22	5-8	8	No symptoms.	Uterus slightly enlarged and softened.	Positive	Pregnant
8	24	4	3	Last period scanty; pain in right lower quadrant.	Enlarged, thickened, palpable appendage on right side; question of extrauterine pregnancy.	Negative	Not pregnant
9	30	4-7	8	Patient believes that she is pregnant.	Cervix soft, uterus normal in size, slight bloody discharge.	Negative	Not pregnant
10	35	4	5	Hot flushes; dizzy spells; periods scanty; nausea and heartburn.	Unsatisfactory; patient not coöperative.	Positive	Pregnant
11	33	8-48	7	Irregular flowing for three months.	Uterus three times larger than normal; ? pregnancy.	Negative	Not pregnant—carcinoma of body of uterus.
12	25	4-7	8	Irregular bleeding since last period.	No definite evidence of pregnancy.	Positive	Pregnant
13	30	Lactation Amenorrhoea		Amenorrhoea since parturition five months ago; nausea and vomiting.	Uterus slightly enlarged.	Negative	Not pregnant
14	?	6-16	7	Nausea and urinary frequency.	? Pregnancy.	Negative	Not pregnant; menstruated four months later.
15	21	4-8	10	Unmarried; nausea and vomiting.	Not done.	Positive	Pregnant
16	23	8-12	?	No symptoms.	No evidence of pregnancy.	Negative	Not pregnant
17	?	3-5	7	Delivered 3 months previously. Period seven weeks ago was the only one since delivery.	? Pregnancy.	Negative	Not pregnant; menstruated nine weeks later.
18	26	Lactation Amenorrhoea		Nausea.	Not done.	Negative	Not pregnant

TABLE OF CASES (*Continued*)

Case No.	Age	Past Menstrual Cycle in Weeks	Interval from Last Menstruation to Test in Weeks	Other Data in History	Physical Examination	Aschheim-Zondek Test	Subsequent Clinical Course
19	36	4	12	Married 16 years; never conceived.	Fibroid uterus, 1½ times normal size.	Positive	Pregnant
20	29	4	8	Patient delivered six months previously. Has had only one period since delivery.	Negative.	Negative	Not pregnant
21	?	4-20	6	Patient has had several miscarriages which she attributes to excessive activity during early pregnancy of which she was not aware because of habitual irregularity.	Unsatisfactory on account of marked obesity.	Negative	Not pregnant
22	27	4	12	?	Uterus slightly enlarged; examination difficult because of obesity.	Negative	Not pregnant
23	22	4-6	9	?	Negative.	Negative	Not pregnant
24	25	4	26	Symptoms of pregnancy began two months after last period.	Uterus slightly enlarged.	Negative	Two months after the test, the patient miscarried twin foetuses, of about three months development, which had been dead for a long time, probably before the time of the Aschheim-Zondek test.
25	21	4	9	Acute gonorrhea.	Acute cervicitis; uterus slightly enlarged and softened.	Negative	Not pregnant
26	38	4	6	Tingling of breasts.	Not done.	Positive	Pregnant
27	36	4	5	No symptoms.	Not done.	Negative	Not pregnant
28	29	4	10	Nausea and vomiting.	Uterus enlarged to size of 2½ months pregnancy.	Positive	Pregnant
29	30	4	9	A few days prior to the test there was a mild flow; ? catamenia.	Not done.	Negative	Not pregnant
30	25	4	6	Stained for three days a week ago.	? enlargement of uterus.	Positive	Pregnant
32	24	4	2	Last menstruation six weeks after the preceding period; nausea and vomiting set in two weeks prior to the last period.	No evidence of pregnancy.	Positive	Pregnant
33	?	?	6-7	?	Suggestive of pregnancy.	Positive	Pregnant
34	25	9	5	Continuous staining since last period.	Inconclusive on account of obesity.	Negative	Not pregnant
36	28	4	5	Slight staining a few days previously.	Uterus considerably enlarged, due to multiple fibroids.	Positive	Pregnant

TABLE OF CASES (*Concluded*)

Case No.	Age	Past Menstrual Cycle in Weeks	Interval from Last Menstruation to Test in Weeks	Other Data in History	Physical Examination	Aschheim-Zondek Test	Subsequent Clinical Course
38	25	8-48	8	Hypomenorrhoea sterility.	Uterus normal in size and position.	Negative	Not pregnant
40	33	12-24	33	No symptoms. Despite definite evidence of pregnancy patient does not believe that she is pregnant.	Uterus enlarged to size of four months pregnancy.	Positive	Pregnant
42	24	2-7	5-6	Nausea.	Uterus normal in size.	Negative	Not pregnant
43	28	4-20	18	Pseudocyesis.	No evidence of pregnancy.	Negative	Not pregnant
44	20	4	8	No symptoms.	Cervix soft; uterus not enlarged.	Positive	Pregnant
45	27	4	6	Believes that she is pregnant.	Cervix soft, uterus somewhat soft but seems of normal size.	Negative	Menstruated one month later.
46	28	4	9	No symptoms.	Probably early pregnancy.	Positive	Pregnant
47	44	4	6-7	No symptoms.	Multiple fibroids of uterus. ? pregnancy.	Negative	Not pregnant
48	?	4-20	8	Aschheim-Zondek test done three months previously was negative. (Case No. 21.)	Unsatisfactory.	Negative	Not pregnant
49	31	4-12	12	No symptoms.	No evidence of pregnancy.	Negative	Not pregnant
50	23	4	8	No symptoms.	No positive signs of pregnancy; secondary anaemia.	Negative	Not pregnant
51	49	4	7	No symptoms.	No positive signs of pregnancy.	Negative	Not pregnant
52	48	4	7	?	?	Negative	Not pregnant
53	35	4-8	12	No symptoms.	Uterus slightly enlarged and firm.	Negative	Not pregnant
54	22	4	8	Unmarried; no symptoms.	Not done.	Positive	Pregnant
55	26	4-24	8	Nausea.	No evidence of pregnancy; endocervicitis.	Negative	Not pregnant
58	35	4	3	Pain in right lower quadrant and slight staining for last few days.	Uterus slightly enlarged; marked tenderness in both vaults but no mass. Lipiodol injection of uterus showed no evidence of intrauterine or extrauterine pregnancy.	Positive	Patient subsequently developed increased pain in the right lower quadrant and a mass became palpable; operation revealed a tubal pregnancy on the right side.
60		4	14	Thyroidectomy one month before last period.	Uterus slightly enlarged but not to correspond with period of amenorrhoea.	Negative	Not pregnant

the mortality of 15% reported by Aschheim and Zondek. After starting to use ether-washed urine, the mortality was reduced to 4%. The incidence of negative reactions in individual mice in positive cases was 10%. This gives an error factor of 14% due to death or non-reactiveness of the mice, which is reduced to .87% by using five mice. This gives mathematical corroboration of Aschheim and Zondek's insistence on the importance of using not less than five mice for each test.

Several cases are worthy of more detailed consideration because they illustrate strikingly the practical value of the test.

CASE No. 2—A woman whose periods were extremely irregular had intermittent staining for two months after a period of amenorrhoea for six months. Her physician was unable to determine whether a pregnancy was present; examination was very difficult on account of a vaginal constriction ring and was followed by profuse bleeding. The Aschheim-Zondek test was positive. Two months later the foetal heart was heard.

CASE No. 15—A girl, unmarried, had amenorrhoea for two months, and complained of nausea and vomiting. Her physician, who knew her intimately, suspected the possibility of pregnancy, but was reluctant to ask her about it, or to examine her pelvically, particularly since her menses were habitually irregular. The Aschheim-Zondek test was positive, and the physician fortified by this result, was able to act accordingly.

CASE No. 24—A woman of 25, whose past menstrual history was normal, had her last period on November 6, 1929. On January 29, 1930, symptoms of pregnancy were present. On June 19, 1930, vaginal examination revealed that the uterus was only slightly enlarged.

The Aschheim-Zondek test done at this time was negative. Two months later the patient miscarried twin foetuses, badly macerated and apparently dead for a long time. The foetuses had attained a development of about three months, which indicates that they probably died in February or March, about three months before the Aschheim-Zondek test was done.

CASE No. 32—A woman of 23, whose periods had previously been regular, had amenorrhoea for six weeks, followed by a normal menstrual period. Nausea and vomiting, which began during the latter weeks of amenorrhoea, persisted, however. There were three factors against a diagnosis of pregnancy: (1) the patient, an intelligent woman, had just completed what she described as a perfectly normal menstrual period; (2) pelvic examination revealed no physical signs of pregnancy; (3) a diagnosis of non-patency of the tubes had been made a few months previously by lipiodol injection. The Aschheim-Zondek test was positive, however, and the diagnosis of pregnancy was later confirmed.

CASE No. 34—A woman with a child one year old consulted her physician on account of continuous flowing of one month's duration. Menstruation was reëstablished six months after the birth of the baby and recurred regularly for four periods. She had amenorrhoea for two months preceding the attack of prolonged bleeding. Pelvic examination was unsatisfactory on account of marked obesity. A diagnostic curettage seemed indicated, but her physician hesitated to do it because of the possibility of an early pregnancy. The Aschheim-Zondek test was negative. A curettage was then done; the pathological diagnosis was hypertrophied endometrium. The periods have been regular since then.

CASE No. 51—A married woman, 35 years of age, who menstruated regularly every twenty-eight days, had her last period on September 8, 1930. On September 19 and September 22 she had attacks of pain in the right lower quadrant and slight vaginal bleeding. On examination, September 22, the uterus was slightly larger than normal and quite tender on attempt to move it; there was also marked tenderness in both vaults, but no mass could be felt. A specimen of urine, taken September 27, nineteen days after the last menstruation, gave a positive Aschheim-Zondek reaction. On October 3, in order to differentiate intrauterine and ectopic pregnancy, a lipiodol injection of the uterus and tubes was done. The roentgenologist reported that he could find no evidence of pregnancy, either intrauterine or tubal. On October 11 there was increased tenderness in the right side and at this time a definite mass could be felt. On operation a cyst of the right ovary was found and an unruptured pregnancy in the fimbriated end of the right tube.

CONCLUSIONS: The Aschheim-Zondek test for pregnancy, carried out in fifty-two clinically obscure cases, was positive in twenty cases, all of which proved to be pregnancy, and negative in thirty-two cases, thirty-one of which proved to be non-pregnancy, and one a blighted ovum. In the light of these findings, and equally accurate results reported by the originators and others, the test must be regarded as an extremely reliable aid to the diagnosis of early pregnancy.

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OUR FALLING BIRTH RATE*

BY GEORGE H. BIGELOW, M.D.,† AND ANGELINE D. HAMBLEN†

THE decreasing birth rate in most of the "civilized" countries has been a matter of frequent comment. In general, the longer a given community has been under the influence of "modern civilization", that is, the "older" it is, the more marked is this decrease. It is, of course, recognized by health officers and all those in any way associated with sociological problems that there is a relationship between the density of a population and such factors as standards of living, sickness, pauperism, and social unrest, though except at the extremes this relationship is usually subtle and subject to dispute. It seems that almost all figures on these matters are open to as diverse interpretation

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as those in regard to the alcohol problem, and that while some receive them with gratification, almost invariably there are others who view them with alarm. This lays anyone presenting any aspect of this matter open to such epithets as Malthusian, Victorian, or worse.

However, with a decrease of 35 per cent. (from 26 per 1000 to 17) in the crude birth rate in Massachusetts from 1900 to 1929 and with the rate of decrease speeding up in the last ten years, it seemed worth while to consider the matter even briefly. Of course, since the war the population distribution of the State has been profoundly affected by the rigid restriction of immigration, though there were racial changes going on before that time. It is well known that the foreign-born coming to this country, largely as young adults, have had consistently a higher birth rate than the native-born. It seemed, then, that it would be interesting and enlightening to know how much of the recent birth rate decrease has been due to a decrease among the foreign-born.

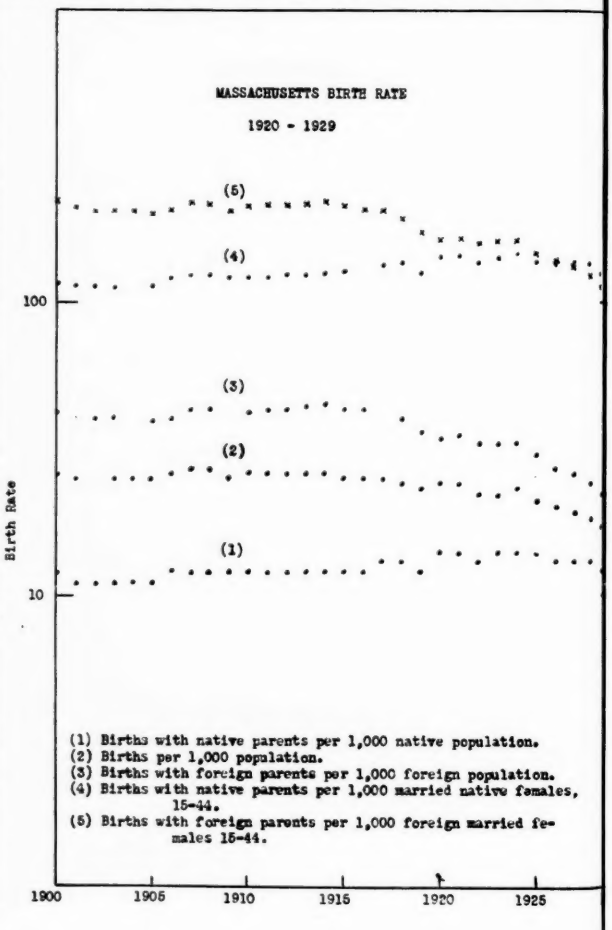
The appended table and chart show the annual birth rate per 1000 of the population by years for the thirty-year period from 1900. Up to 1917 the rate was essentially constant. Since 1924 there has been a consistent decrease.

Where both parents were foreign-born, the data show a fairly flat rate up to 1917, fluctuating from 39 to 45 per 1000 of the foreign-born population. Since then there has been a steady decrease to one-half that figure. This phenomenon is also apparent if we use the rate per 1000 married foreign-born females, age 15 to 44. Here the rate drops 45 per cent. from 1917 to 1929. The explanation of this very large decrease is not entirely clear. It may be in part because, during this period, a larger proportion of the foreign-born women are each year passing beyond the child-bearing age. Another contributing factor is undoubtedly the decreasing marriage rate among foreign-born women, though this would not affect the latter figure. In 1900 the marriage rate was 13 per 1000 foreign-born, in 1920 this had dropped to 11, and by 1929 to 8. The last part of this is probably further evidence of the phenomenon mentioned above, that with restricted immigration a larger proportion of the foreign-born women are passing out of the age of child-bearing and

MASSACHUSETTS BIRTH RATE*

Year	Crude Birth Rate. Number of Births per 1,000 population.	Births With Native Parents per 1,000 Native Population.	Births With Foreign Parents per 1,000 Foreign Population.	Births With Native Parents per 1,000 Native Married Females. 15-44.	Births With Foreign Parents per 1,000 Foreign Married Females. 15-44.
1900	26	12	42	119	225
1901	25	11	41	115	214
1902	25	11	40	115	208
1903	25	11	40	114	208
1904	25	11	40	115	206
1905	25	11	39	115	200
1906	26	12	40	123	207
1907	27	12	43	126	220
1908	27	12	43	126	219
1909	25	12	41	122	208
1910	26	12	42	122	212
1911	26	12	43	122	216
1912	26	12	43	124	215
1913	26	12	44	126	218
1914	26	12	45	126	221
1915	25	12	43	129	213
1916	25	12	43	129	209
1917	25	13	42	135	207
1918	24	13	40	139	195
1919	23	12	36	127	174
1920	24	14	34	144	165
1921	24	14	35	144	168
1922	22	13	33	138	160
1923	22	14	33	142	161
1924	23	14	33	148	162
1925	21	14	30	140	149
1926	20	13	27	138	140
1927	19	13	26	139	134
1928	18	13	24	136	124
1929	17	12	22	128	113

*At the time of computing these rates the number of native and foreign born in the population of Massachusetts according to the 1930 census had not been published. It was assumed that the native population increased at the same rate from 1920 to 1929 as it did from 1910 to 1920. In the case of the foreign population an estimate was made based on the findings in surveys of certain cities and towns. The estimate was felt to be very conservative and the true rate has probably dropped even more abruptly than the rate given here.



also of high marriage rates. A third factor may be that, as the average length of residence of these women in this country increases, they are more and more adopting the standards of living of their native-born sisters. This is suggested by the birth rate where both parents were foreign-born per 1000 married foreign white females, age 15 to 44. In 1900 the rate was 225, while in 1920 it had fallen to 165.

Where both parents were native-born, the data show essentially a constant rate over the thirty years with a tendency to a 10 per cent. rise during the last fourteen years, when the rate per 1000 of the native-born is considered. However, if we use the rate per 1000 of the married native-born females age 15 to 44, this rise becomes even more apparent. To 1917 the curve is essentially flat, but from then on there is a definite increase. How much of this increase is due to the first generation in this country of foreign-born parents (included in the census as native-born) cannot be readily estimated. However, it is obvious that over the years this first generation of foreign-born must be of increasing importance and may well contribute to the apparent increase in the native-born birth rate. This and other matters dealt with in this paper are discussed in a pamphlet which has recently come to hand* from the Brookings Institution.

CONCLUSIONS

The steady decrease in the birth rate in Massachusetts which has occurred since 1917 has been due entirely to a decrease in the rate among foreign-born. Indeed, during this time there has been a slight, but definite, increase in this rate among the native-born. From this we might expect that as the proportion of foreign-born continues to decrease, the birth rate should tend to stabilize and even perhaps show a slight gradual increase unless some other factors, such as economics, interfere with this complex phenomenon.

It is realized that population estimates from 1921 to 1929 are subject to great error due to restricted immigration, and as a result, the birth rates appended are necessarily not accurate; but as the rates for the census years 1900, 1910 and 1920 show the same general trends it would appear that the conclusions are sound.

* "The Fecundity of Native and Foreign-Born Women in New England", Joseph J. Spengler, Pamphlet Series Vol. II, No. 1, June 30, 1930, Brookings Institution, Washington.

ARE VITAMIN D AND IRRADIATED ERGOSTEROL IDENTICAL?*

BY ARTHUR D. HOLMES, M.D.†

THE DISCOVERY OF VITAMIN D

VITAMIN D, as such, was first recognized by McCollum[1] early in 1922. McCollum noticed that when cod liver oil was subjected to atmospheric oxidation, its vitamin A potency was diminished, more or less, in proportion to the intensity of the oxidation that had taken place. However, the cod liver oil, which had been subjected to oxidation, seemed to retain value for curing rickets. Accordingly, he concluded that cod liver oil contained two vitamins—an antiophthalmic vitamin and an antirachitic vitamin. His opinion in this regard was not immediately accepted by all investigators. Eventually, however, the results of his studies and those of investigations conducted by numerous workers seemed to show conclusively that cod liver oil contained two fat-soluble vitamins. These have been designated "vitamin A" and "vitamin D". A number of other substances have been shown to contain vitamin D, but cod liver oil is apparently the richest, naturally occurring source of this vitamin.

ORIGIN OF IRRADIATED ERGOSTEROL

Hess[2] and Steenbock[3] independently showed that some food materials, when subjected to the action of ultraviolet light, acquired antirachitic activity. In subsequent studies by these and other investigators, a wide variety of materials were subjected to ultraviolet irradiation; some became antirachitic and others did not. Obviously, a question soon arose concerning the nature of the substance which acquired the antirachitic activity. It was early observed that this activity was acquired by materials that contained fat and it was noted that this substance resided in the unsaponifiable portion of the fat. Since the unsaponifiable portion of fat contains a large proportion of cholesterol, it was thought, at first, that cholesterol was the substance which acquired the antirachitic activity. This theory was apparently substantiated when it was found[4] that supposedly pure cholesterol became antirachitic when exposed to ultraviolet radiation. The results of further studies seemed to indicate that the cholesterol underwent some change or rearrangement at the site of the double bond, since cholesterol became antirachitic and dihydroxycholesterol did not become antirachitic when subjected to ultraviolet irradiation. Subsequently, the brilliant investigations of Bills[5], Heilbron[6], Hess[7], Rosenheim[8], Steenbock[9], Webster[10], and Windaus[11] showed that it was not the cholesterol but ergosterol, which was associated with it, that became antirachitic when subjected to ultraviolet radiation.

NATURE OF IRRADIATED ERGOSTEROL

As soon as it had been definitely established that ergosterol was the substance which became

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antirachitically active when irradiated, biochemists and clinicians visualized the possibility of preparing a synthetic antirachitic product for therapeutic use, and so the research of ergosterol-containing substances began. Ergosterol was originally separated from ergot, but it is not feasible to prepare it from this source commercially, due to the high cost. Consequently, numerous substances were tested for their ergosterol content, but while it was found in numerous substances, yeast appeared to be the most desirable source for commercial purposes. However, Bills, Massengale and Prickett[12] have submitted evidence that different species of yeast vary enormously in ergosterol content. Accordingly, it was first necessary to develop a strain of yeast having a high ergosterol content. The preparation of ergosterol from yeast is tedious and expensive. Purified ergosterol is a white crystalline substance. When it is dissolved in various solvents and subjected to ultraviolet irradiation, it acquires antirachitic activity hundreds of times greater than that of any other substance. On account of this excessive antirachitic activity, irradiated ergosterol is diluted with corn oil, which also serves to protect it from deterioration. In this form it is known in the trade under the name of "Viosterol."

VITAMIN ACTIVITY OF VITAMIN D AND IRRADIATED ERGOSTEROL ASSUMED TO BE SIMILAR

In numerous investigations of the antirachitic activity of irradiated ergosterol, laboratory animals were cured of induced rickets. It was also found in clinical studies that when irradiated ergosterol was administered as a prophylactic or curative agent for rickets, the condition was alleviated. Cod liver oil used as a source of vitamin D also produced the same results. Naturally, then, irradiated ergosterol was considered by many as synthetic vitamin D. As a consequence, the terms vitamin D and irradiated ergosterol have been very frequently used interchangeably. Of late, however, some evidence has accumulated which raises a question as to whether vitamin D, as it occurs naturally in such substances as cod liver oil, is identical with irradiated ergosterol.

RECENT ANIMAL EXPERIMENTS DESIGNED TO COMPARE NATURAL VITAMIN D AND IRRADIATED ERGOSTEROL

Hess and Supplee[13], in a study of the relative antirachitic activity of ultraviolet light, of irradiated ergosterol, and of cod liver oil, found that "whereas chickens are regularly protected against leg weakness by an addition of 1% of cod liver oil to their ration, a supplement of irradiated ergosterol equivalent to many times this amount failed to afford protection". They also found that the bone ash of the chicks receiving irradiated ergosterol was comparatively low.

Massengale and Nussmeier[14] fed White Leghorn chickens, from hatching until ten weeks of age, a ration with a high mineral content and well provided with satisfactory proteins. The chicks were divided into comparable pens all of which received the same basal ration. One pen served as a control pen. Other pens received as supplements to the basal ration, 2% cod liver oil, and amounts of irradiated ergosterol equivalent in antirachitic value to 2%, 20%, 200% of cod liver oil. Judged by the ash content of the femur removed from the chicks at ten weeks of age, cod liver oil possessed sufficient antirachitic value to produce normal bone development. The irradiated ergosterol was decidedly inferior as an antirachitic agent for the authors report that "it was necessary to administer a quantity of activated ergosterol equivalent, by assay on rats, to 200 per cent. of cod liver oil, to produce the effect of 2 per cent. of cod liver oil".

Mussehl and Ackerson[15] fed chicks for the first eight weeks of life a ration complete except for the antirachitic factor. Cod liver oil, irradiated ergosterol, or irradiated yeast was used as a source of the antirachitic factor for supplementing the basal ration. One pen received 2% cod liver oil. Irradiated ergosterol, theoretically equivalent to 2%, 4%, 6%, 20%, 30% and 50% of cod liver oil, and irradiated yeast, theoretically equivalent to 10% cod liver oil, were used as a supplement of the ration of other pens. The authors conclude: "Chicks reared on our basal ration supplemented with two per cent. of cod liver oil made excellent growth with no evidence of rickets. When irradiated ergosterol was added to the same basal ration in amounts theoretically equivalent to 2, 4, 6, 20, and 50 per cent. of cod liver oil respectively, growth was subnormal and the physical appearance of the chicks indicated rickets."

The general conclusions to be drawn from the above noted investigations is that irradiated ergosterol is decidedly inferior to cod liver oil in curing avian rickets. This conclusion apparently raises a question as to whether the experimental conditions and experimental rations employed in chick tests differ enough from those of rat tests to produce different results, or whether vitamin D, as it occurs in cod liver oil, differs radically from irradiated ergosterol in its action as a therapeutic agent for the treatment of rickets.

RECENT CLINICAL STUDIES CONCERNING EFFICACY OF NATURAL VITAMIN D AND IRRADIATED ERGOSTEROL

Barnes, Brady, and James[16] carried on a carefully controlled investigation to determine the relative value of irradiated ergosterol and cod liver oil as a prophylactic antirachitic agent when ingested in equivalent antirachitic doses. No attempt was made to select cases and the 216

cases which were included in the study were assigned in rotation to one of four groups. The experiment was continued from December to April inclusive. Since 98% of the subjects were well or benefited at the end of the experiment the authors concluded that the administration of three teaspoonfuls of cod liver oil daily (1400 vitamin D rat units) contained an adequate supply of the antirachitic agent to be effective as a prophylactic for rickets. The second group received three teaspoonfuls of cod liver oil daily, fortified with sufficient irradiated ergosterol to increase the antirachitic value to 3750 rat units —2½ times the antirachitic potency of the cod liver oil group. The authors state: "There was no evidence of superiority among this group as compared with the group given cod liver oil." The third group received irradiated ergosterol at the rate of 1250 rat units per day. The irradiated ergosterol served to prevent or cure rickets in only 44% of the cases. The authors state: "The results in this group were not significantly better than those for the control group of untreated subjects." The results of this investigation, briefly stated, were: 98% of the subjects in the cod liver oil group (1400 rat units daily) were well or benefited at the end of the experiment, whereas only 44% of the subjects in the irradiated ergosterol group (1250 rat units daily) were well or benefited at the end of the experiment.

Hess, Weinstock, and Rivkin[17], in an extended series of observations, found "the action of two specific antirachitic agents (irradiated ergosterol and cod liver oil) is likewise not identical in relation to infantile rickets". They found that "6 teaspoonfuls of cod liver oil brought about cure where 20 drops of irradiated ergosterol (supposed to be the equivalent of 10 teaspoonfuls of cod liver oil) failed to afford complete protection". In one instance in which mild rickets developed in spite of the administration of irradiated ergosterol, 6 teaspoonfuls of cod liver oil affected healing in a few weeks.

De Sanctis and Craig[18] have also conducted a lengthy study to determine the relative prophylactic value of cod liver oil and irradiated ergosterol. One hundred infants were given cod liver oil and 123 irradiated ergosterol as a prophylactic for rickets. The antirachitic potency of the cod liver oil and the irradiated ergosterol were determined as Steenbock rat units. The cod liver oil group received 3 teaspoonfuls of oil daily (or from 140 to 170 Steenbock rat units). The irradiated ergosterol group received 10 drops daily of viosterol 100 D (or 300 Steenbock rat units). Thus the antirachitic value of the irradiated ergosterol was about twice that of the cod liver oil. In the cod liver oil group, rickets was prevented in 97% of the cases. In the irradiated ergosterol group, rickets was prevented in only 77% of the cases. The authors concluded that "from the foregoing observations, one of two conclusions may be drawn: either the present recommended prophylactic dose of viosterol is too small to prevent rickets or rickets is not due to a deficiency of vitamin D alone. We are inclined to believe that the second of these possibilities is true".

The results of the foregoing investigations offer quite definite evidence to show that cod liver oil and irradiated ergosterol, administered on the basis of equivalent antirachitic value, do not produce similar results. As judged by the evidence noted above, cod liver oil appears to be greatly superior to irradiated ergosterol as a prophylactic agent for rickets. In attempting to explain this situation, one may question whether vitamin D, as it occurs in cod liver oil, contains some substance or substances therapeutically efficient in the treatment of rickets which are not contained in irradiated ergosterol, or whether the vitamin A present in cod liver oil, but absent from irradiated ergosterol, in some way supplements the therapeutic activity[19] of the antirachitic value of cod liver oil.

RECENT CHEMICAL STUDIES INDICATE DIFFERENCE BETWEEN VITAMIN D AND IRRADIATED ERGOSTEROL

The early studies of the chemistry of irradiated ergosterol indicated that this material was quite similar chemically to vitamin D. Both were found to be soluble in fat or oil; both are destroyed by oxidation; they are not destroyed by saponification; and both lose their antirachitic value when subjected to prolonged irradiation.

Recently Bills, who is the foremost worker in this field in this country, has accumulated a wealth of data concerning the chemistry of ergosterol. In connection with his extensive studies[20] of ergosterol and its esters, he has devoted considerable attention to the question of isomerization. He has collected evidence to show that there are many isoergosterols and that certain isoergosterols are intertransformable. Bills[21] is of the opinion that "these isomeric mixtures may differ in kind as well as in proportion, some components being mutually transformable with other, and some not". Accordingly, it seems logical that from this accumulated data concerning the chemistry of irradiated ergosterol, he should conclude that "activated ergosterol and cod liver oil are not identical in antirachitic action. Comparison of these two agents involves a consideration of isomerism".

DISCUSSION

In the above-noted investigations, the antirachitic activity of vitamin D, as it occurs in cod liver oil, was compared with that of irradiated ergosterol. The data which have been assembled were obtained in experiments with laboratory animals, in clinical studies, and in laboratory studies of the chemical nature of ir-

radiated ergosterol. These data seem to indicate that, for the treatment of rickets, either vitamin D as it occurs in cod liver oil possesses properties not present in irradiated ergosterol, or that the vitamin A or some other substance in cod liver oil enhances the antirachitic vitamin in the oil so that it is more effective in the treatment of rickets than equivalent rat units of irradiated ergosterol. In view of the constantly changing status of irradiated ergosterol as a therapeutic agent, it is obviously wise to delay final conclusions in this regard until additional data have been accumulated. In the meantime, it would appear that referring to vitamin D and irradiated ergosterol as synonymous is more likely to confuse than to clarify the situation. Accordingly, it would seem the part of wisdom to refrain from referring to vitamin D and irradiated ergosterol as though they were identical substances.

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PERFORATION OF THE INTESTINE BY A FISHBONE*

BY THEODORE S. BACON, M.D.,† AND PHILIP GORDON CORLISS, M.D.†

THE following case is of interest because of the difficulty which it presented of making the correct diagnosis, and the unusual manner in which death was caused:

The patient was an English widow of sixty-six, who entered the Springfield (Mass.) Hospital as a medical ward patient on August 20, 1930, complaining of gas and epigastric distress after eating, of more than fifteen years' duration, worse in the past few weeks. She seemed mentally confused and her memory was poor, but the following history was obtained:

She had been subject to indigestion for a great many years. Her husband had died ten months previous to her admission, and she had been in poor circumstances since his death. About four weeks before admission her abdomen began to swell. She frequently vomited bile and food, but never any blood. She had no pain or any burning sensation. She felt comfortable when lying down. Liquid food upset her as much as solid food; sometimes she vomited after a small drink of water. For four weeks she had vomited early every morning, and again whenever she ate. Her bowels had always been loose or regular except that during the past winter she had had a spell of constipation. For four weeks previous to admission she had had five or six loose movements a day, never passing any blood, but with occasional bright green or yellow stools.

The rest of the history was essentially negative. She had had maldevelopment of her sexual organs, and had never menstruated, although her married life seemed to have been a happy one. She had had erysipelas twice, followed the last time by "rheumatism", which had also followed frequent colds. She had had typhoid fever and diphtheria.

On admission her temperature was subnormal; the pulse was eighty-eight and of poor quality; the respirations, twenty-two. Physical examination showed a sweet-mannered English lady, very obese, lying on her right side in bed. The following findings were of interest: She had six very poor lower teeth; no uppers. There was a soft systolic heart murmur, best heard at the apex. The abdomen was hugely dis-

*Springfield (Mass.) Hospital.

†Bacon—Visiting Physician, Springfield Hospital. Corliss—House Officer, Springfield Hospital. For records and addresses of authors see "This Week's Issue", page 223.

tended, egg-shaped, with the apex on the right side of the midline. To the majority of observers there was, definitely, fluid in the abdomen, probably encapsulated, and more on the right side. Vaginal examination revealed a small introitus and a vagina which would barely admit the tip of the finger; no cervix could be felt.

The urine was amber colored, clear, acid; the specific gravity 1018; it showed no sugar; the slightest possible trace of albumen, ammonium urate crystals, eight to ten white blood corpuscles, bacteria, and a rare hyaline cast. The blood findings were: hemoglobin, 90% (Talìqvist); white blood corpuscles, 7,000; Wassermann test negative.

The Medical Service, feeling that the condition was surgical, possibly an ovarian cyst, transferred the patient to the Surgical Service on August 22. The surgeons made a provisional diagnosis of ascites, probably due to carcinoma of the ovary. The abdomen was tapped, but only a few cubic centimeters of bloody fluid were obtained. The patient was carried on the surgical service, and was given subpectoral fluids in the hope that her condition would improve sufficiently to warrant an exploratory operation.

On September 2 she was returned to the medical service, practically moribund. Her temperature had stayed below 100° by rectum; her pulse had varied between 60 and 140. Her respirations rose from 20 to 40 and she developed hypostatic pneumonia.

Digifoline was given. A pressure sore developed over her right hip. The patient became incontinent, comatose, the respirations very labored, and she died the night of September 5.

A postmortem examination was made the next morning. About two quarts of serosanguineous fluid were found in the abdominal cavity. The intestines were matted together by adhesions into a gangrenous ball. The peritoneum showed signs of inflammation in the left lower quadrant. There were no glands or other signs of malignancy. On freeing the sigmoid, which was covered with inflammatory exudate, a fishbone was seen perforating halfway through the intestinal wall. It was about three-fourths of an inch long.

This case recalled memories of another recent case in this hospital in which the preoperative diagnosis was acute appendicitis. The appendix appeared perfectly normal; on exploring the abdomen the omentum was found curled up in a ball; in its center was a fishbone.

SUMMARY

Case of death following perforation of sigmoid by a fishbone, causing peritonitis, adhesions, and gangrene of the intestine.

MORTALITY FROM INFLUENZA AND PNEUMONIA IN 50 LARGE CITIES OF THE UNITED STATES, 1910-1929

A report recently issued by the United States Public Health Service deals with influenza and pneumonia mortality in the 50 cities of the United States that had 100,000 or more in population in 1910. For 35 of these cities, with an aggregate population of nearly 25,000,000, the number of deaths from influenza and pneumonia by weeks is available from September, 1918, to the present time. Deaths for the country as a whole are not available by weeks, and so these data for this large group of cities have considerable significance. To supplement them, monthly influenza and pneumonia death rates have been computed for the same cities for the years 1910-18.

Since 1915 there have occurred ten distinct periods, each of 8 to 31 weeks' duration, in which the mortality from influenza and pneumonia in this group of widely dispersed cities was so greatly increased as to denote epidemic conditions. Minor epidemics prior to the 1918 pandemic occurred in January, 1916, January, 1917, and April, 1918, with a very slight rise in the mortality in April, 1915, also. We are accustomed to think of the epidemic of 1918 as occurring in the fall, and it is true that the enormous peak, which occurred in the middle of October, overshadowed all prior and subsequent recorded epidemics. In addition to this tremendous peak, the epidemic stretched out over a period of 31 weeks, from September 15, 1918, to April 19, 1919, and even as late as the latter part of January, 1920, the mortality from influenza and pneumonia in excess of the expected rates for that season of the year was greater than the excess mortality during the epidemic of 1929. In the early months of 1920 a very sharp epidemic occurred, with excess mortality greater than during any other epidemic since the 1918 epidemic. In February, 1922, February, 1923, March, 1926, and January, 1929, other epidemics occurred, and in May of 1928 there was a slight rise in the influenza mortality which extended to many sections of the country. The combined excess mortality from influenza and pneumonia during these six epidemics that have occurred since the pandemic of 1918 was only about one-half of that of 1918-19.

In addition to the data for the group of cities, this report contains monthly excess death rates for each of the 50 cities throughout the period 1910-29 and weekly excess death rates for a large number of the cities during the three major epidemics, 1918-19, 1920, and 1928-29. In every one of the various epidemics, even including the pandemic of 1918-19, there is great variation in the severity of the different epidemics in different cities. Moreover, there are periods of excess mortality from influenza and pneumonia in certain cities and sections of the country which are not of sufficient importance to show up in the combined data for the country as a whole.

The report is intended as a rather detailed history of influenza and pneumonia mortality during the past twenty years as a background for the consideration of the present situation with respect to the respiratory diseases.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 17051

THREE WEEKS' PAIN IN THE RIGHT WRIST

MEDICAL DEPARTMENT

A Canadian soap worker thirty-eight years old entered November 7 from the Emergency Ward complaining of pain in the right wrist of five weeks' duration.

During the past four months he had had five or six sudden attacks of a feeling of pressure in the left thorax, usually occurring after meals and lasting about an hour, accompanied by profuse sweating. At times they were relieved by tea. Except for these attacks he felt very well until September 29, when he found that using a wrench caused pain in his right wrist. He continued to work for ten days with his right arm in a sling. The wrist rapidly became swollen and hot. A physician treated him for arthritis. Two weeks after the onset he began to have severe frontal headaches with pains all over his body. By this time his wrist had become normal. Two weeks before admission his physician told him he should stay in bed and gave him pills which caused frequent urination. The patient remained at home, feeling somewhat weak. A week before admission he stopped taking the pills given by his physician. He at once developed severe orthopnea, dyspnea, a frequent choking and pressing sensation in midchest which had remained severe and swelling of the ankles which had been much more marked in the past two days. Throughout the illness he had had moderate cough and had occasionally raised some material which he said felt "crackling" between his fingers. Recently he had noticed that he could see the pulse jump in the vessels of his arms. Recently his bowels had been constipated. The day before admission he passed stools with some black discoloration.

Records of the Outpatient Department show that on August 5, three months before admission to the wards, he came complaining of pain in the umbilical region of six days' duration. He said that the pain in the midchest following meals had been relieved by soda. He gave a history of swelling of the ankles for three days a year before this followed by swelling of the wrists and elbows for three days. He had noticed this since that time. He had had some morning cough. The morning of the visit the sputum was blood-streaked.

Examination showed a rash on the neck, some enlargement of the cervical glands, a mid-diastolic and a rumbling presystolic murmur, clubbing of the fingers. A Hinton test was negative.

His family history is good. His wife's first pregnancy terminated in a miscarriage at three months. One child was living and well.

He had a severe attack of rheumatic fever at ten years lasting two weeks. He also had scarlet fever in childhood. He was gassed with chlorine in 1918. After four months in a hospital he was supposed to be cured.

He gave his history with some confusion and inconsistency.

Clinical examination showed an obviously ill man propped up in bed breathing with effort in short gasps. The lips and mucous membranes were pale but not very cyanotic. The sclerae were slightly suffused. The skin showed wasting of the subcutaneous tissues. The lips showed sordes, the teeth slight caries. The apex impulse of the heart was diffuse in the anterior axillary line in the fifth and sixth interspaces. The supracardiac dullness was 5 centimeters. There was a diastolic thrill over the sternum and along its left margin. The action was rapid. The sounds were of poor quality. There was a loud rasping diastolic murmur over the entire precordium. The first sound was faint. In the axilla the two sounds were better distinguished. An electrocardiogram showed normal rhythm, rate 90, low voltage, flat T waves in all leads. The pulses were equal, regular, synchronous and collapsing. There was loud pistol shot over the femorals and brachials. The systolic blood pressure at admission was 210, the diastolic undetermined. Later the systolic was questioned at 285 or 200; the diastolic was 25. The lungs showed diminished breath sounds with moist crepitant râles throughout both backs. The sternum was depressed. The cardiac dullness was made out with finger percussion because the whole chest was hyperresonant. The liver flatness extended to the iliac crest, where a tender edge was easily felt. There was dullness in both flanks. There was secondary osteoarthropathy of the fingers and toes. The legs and ankles showed pitting edema. The pupils and fundi were normal. The knee jerks and ankle jerks were increased. The amount of urine was 90 ounces on one occasion, otherwise normal. The specific gravity was 1.024 to 1.040. The urine was red or brown on three of six examinations, and showed a trace to a large trace of albumin at all examinations, 2 to 20 leukocytes at all, 7 to 20 red cells at the last three, occasional to many granular casts at all. The leukocyte count was 10,200 to 5,900. Two counts showed 69 to 71 per cent polynuclears. A third showed 12 per cent polynuclears, 25 per

cent lymphocytes and 63 per cent immature polynuclears and large mononuclear granulocytes. Two smears showed no abnormalities. The hemoglobin was 80 per cent, the red count 5,220,000. A Hinton test was negative. The non-protein nitrogen was 46.

The temperature was 95.7° to 100.5° with a rise to 103°-105.5° in the last four days. The pulse was 93 to 121. The respirations were 18 to 35.

On November 10 there was a questionable rash on the chest. A cardiac consultant noted a peculiar groaning quality to the diastolic murmur. On January 15 there was Cheyne-Stokes breathing in the morning. That evening and the following day the patient was somewhat irrational. The temperature rose to 103°-104°, the respiratory rate to 35. There was pitting edema of the trunk and forearms and marked ascites. November 18 it was necessary to place him on constant drainage because of retention. He had marked edema of the legs and thighs up to the waist. The left leg and thigh were reddened and tender in the middle third. Pulsations were present and equally weak in both femorals. A surgical consultant found both popliteal pulses easily palpable and the legs hot to the ankles. Below that there was discoloration, blistering and coolness of the feet. He found a similar process starting over the left trochanter. There was no history of sudden onset.

On November 19 the temperature rose to 105° and the patient died.

Clinical Discussion

by Richard C. Cabot, M.D.

Notes on the History

That is quite an extraordinarily acute onset of cardiac symptoms. One does not often see them come on within a week in that way.

The visible jumping of the pulses of course suggests aortic regurgitation.

The black discoloration of the stools may have been due to blood; it may have been nothing.

The suggestion of the history is for rheumatic heart disease with mitral stenosis. The question is, is there something acute on top of it?

Notes on the Physical Examination

We do not expect a loud rasping diastolic over the entire precordium in mitral stenosis alone. We are looking for something more than mitral stenosis when we have that.

I suppose the faint first sound was that at the aortic area. That is the one we are ordinarily interested in in such a case.

When the diastolic blood pressure is undetermined it generally means that it is pretty low. We can say we have first-rate evidence, then, of aortic regurgitation.

Depression of the sternum is of no importance, I suppose.

He had secondary osteoarthropathy such as goes with clubbed fingers. It merely means that the clubbing had been going on long enough to make changes in the bones and tissues.

The urine report makes us suspect the possibility of acute glomerular nephritis.

The differential count of 63 per cent immature polynuclears shows that we have an enthusiast there. When there is infection the polynuclears get younger. That is a fact established by Arneth many years ago.

The non-protein nitrogen of 46 is one of those borderline readings that bother us.

Pulsation was equal though weak in both femorals. There was no femoral block, then.

Differential Diagnosis

Two things seem to be perfectly clear here; first a chronic heart lesion, second, something more than a chronic heart lesion. He has a chronic heart lesion because he has clubbed fingers and osteoarthropathy in the joints. Presumably that is mitral stenosis going back to the age of ten years. But he certainly has something more because of the tremendous pulse pressure and jumping arteries. He certainly now has aortic regurgitation with a very big heart, fever, rather little in the way of blood changes, suggestions of a rash but no petechiae or skin emboli. In the leg there is a definite suggestion of venous thrombosis or possibly a low arterial thrombosis. We have to say subacute bacterial endocarditis on top of an old rheumatic process, presumably aortic, presumably destroying enough of the aortic valve to give us aortic regurgitation, but certainly acute and not merely fibrous or fibrocalcareous. Aside from that I do not see any good evidence. If we have thrombi forming in the peripheral leg veins or arteries there are probably infarcts of the spleen or kidneys and possibly these urinary changes may be due to embolism. But this is a disease that is often associated with acute glomerular nephritis. It is natural therefore to speak of that. There is no evidence that they took a blood culture but it is quite possible.

A Student: Isn't that a very large liver?

Dr. Cabot: No. With a failing heart it is simply a part of the clinical picture. His liver is large, but not extraordinarily large for passive congestion.

A Student: Does one get such kidney symptoms on the basis of congestion?

Dr. Cabot: Yes, though I think it is rather more probable that there are some infarcts or glomerular nephritis or both.

A Student: Is absence of pain very much against infarct of the kidney?

Dr. Cabot: No. We often have kidney infarcts with no pain.

A STUDENT: Could he have had small coronary infarcts to explain the chest pain?

DR. CABOT: The more we know of coronary infarcts the more we find they sometimes occur without any pain at all, as is shown by cases proved at autopsy. That is perfectly possible with or without pain.

A STUDENT: Could this be another attack of rheumatic fever?

DR. CABOT: The organism of rheumatic fever is not the same as the organism of subacute bacterial endocarditis. But it is a very familiar fact that it is those hearts that are already injured by previous rheumatism which get the infection of subacute bacterial endocarditis. Dr. Mallory will tell us how nearly or distantly the organisms are probably related. We know the organism of subacute bacterial endocarditis, but not that of rheumatism.

DR. TRACY B. MALLORY: Was there a positive blood culture during life?

MISS PAINTER: Cultures taken November 9 and 16 showed no growth in five flasks. Cultures taken November 18 were contaminated.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Rheumatic heart disease with mitral and aortic stenosis and insufficiency.
Thrombophlebitis, both lower legs.
Subacute bacterial endocarditis?

DR. RICHARD C. CABOT'S DIAGNOSES

Chronic rheumatic endocarditis of the mitral and aortic valves.
Subacute bacterial endocarditis with aortic regurgitation.
Possibly acute glomerular nephritis.
Possibly infarcts of the spleen and kidneys.
Venous thrombi in the leg veins.

ANATOMIC DIAGNOSES

1. *Primary diseases.*

Chronic rheumatic endocarditis; aortic regurgitation.
Subacute bacterial endocarditis.

2. *Secondary or terminal lesions.*

Thrombophlebitis of the lower extremities.
Multiple infarcts of the lungs.
Acute glomerular nephritis.

PATHOLOGIC DISCUSSION

DR. MALLORY: This man had an old rheumatic heart of course, and a typical bacterial endocarditis on top of it. There was very little deformity of the mitral valve but a very marked one of the aortic. There were vegetations present not only on the aortic valve but also on the wall of the aorta above the valve. He had thrombi in the leg veins and infarcts in the lungs, evidently the result of embolism from them. He had infarcts in his kidney and also the focal type of glomerular nephritis characteristic of cases of bacterial endocarditis. The liver was enlarged but not very strikingly big. We measured the edge as reaching 2 centimeters below the costal margin. A possible explanation of the discrepancy is that a cardiac patient as sick as this is usually examined in an upright position, allowing the liver to sag downward and seem much larger than when the patient is lying flat.

A blood culture taken after death showed no growth.

CASE 17052

PERSISTENT INDEFINITE PAIN IN VARIOUS LOCATIONS

SURGICAL DEPARTMENT

First admission. A Canadian mechanic fifty years old was sent in from the Emergency Ward March 27, a year and eight months before his second admission, complaining of pain in the sternum. At x-ray examination while he was in the Emergency Ward no fracture could be demonstrated.

Records of the hospital show that he had had treatment for the nose at the Eye and Ear Infirmary sixteen years before admission and treatment for gonorrhea in the Urologic Clinic for nine years ending five years before admission. Seven years before admission he was seen in the Orthopedic Clinic for pain in the left shoulder radiating across the chest wall to the opposite side. Five years before admission he was examined in the Male Medical Department for possible gastric disease. X-ray examination showed no evidence of organic disease in the stomach or duodenum. In February, a month before admission he was treated in the Male Medical Department for strain in the pectoral muscles.

Clinical examination showed a well nourished man, apparently in good general condition, with no gross abnormalities except some carious teeth and tenderness along the whole sternum radiating down the left costal margin to the midaxilla. No tumor or swelling was made out. The sternum and ribs felt perfectly normal. The patient could sit up only with difficulty because of pain in the sternum. He stood and walked normally.

The urine was normal. No Bence-Jones bodies were found. The blood is not recorded. A Wassermann test was negative.

The day of admission the temperature was 100°. Afterwards the chart was normal before operation.

X-ray examination showed generalized increased radiability of all the bones taken, including the ribs, clavicles and lumbar vertebrae, characterized by numerous small rounded

punched-out areas of diminished density measuring from 2 millimeters to 1 centimeter in diameter. Examination of the lungs showed the diaphragm to be normal in outline and limited in excursion. There was generalized prominence of the larger lung markings and slight tortuosity of the aorta. Examination three days later confirmed the previous findings. The films of both shoulder girdles showed that the process had involved the upper ends of the humeri and both scapulae. Anterior-posterior and lateral views of the skull showed a number of minute areas of diminished density, but these might have occurred normally in the skull and were not definitely pathologic. There was no evidence of involvement of the hands, forearms or legs. There was an area of periosteal thickening on the medial surface of the midshaft of the right femur. No gross abnormality was noted in the lateral view of the lumbar spine.

A urological consultant found nothing abnormal in the genito-urinary tract.

April 3 three inches of the left eighth rib in the midaxillary line was removed for biopsy. A pathologic report of myeloma was returned.

April 7 the patient was discharged.

History of interval. He continued to have some discomfort in his chest and visited the Tumor Clinic. He had x-ray treatment from May 6 to August 1. There was no progression in the process while he was under treatment.

Second admission. November 6, a year and seven months after his discharge, he reëntered for x-ray treatment.

After admission to the ward, while walking along the corridor he set one crutch on a safety pin. The crutch slipped 8 or 10 inches until stopped by the wall. The patient did not fall, but in an effort to regain his balance jerked his head sharply to one side. He felt a grating in his neck and immediately had excruciating pain in the back of the neck radiating up and down, lasting two or three minutes, then easing. A month before admission he first had similar but much milder pain in the neck on getting up quickly.

Only a superficial physical examination was made. It showed a lean old man lying flat and holding his head motionless, complaining of great pain in the back of his neck. The skin was tanned. There were small bilateral axillary and inguinal glands. The heart was slightly enlarged to the left. The action was slow, the rhythm regular. There was a systolic blow at the apex. The radial arteries were palpable. There was dullness in the left infraclavicular region with no other signs. The abdomen, extremities, pupils and reflexes were normal. There was a small non-tender red lump in the region of the third cervical vertebra.

The urine showed a very slight trace of albumin at two of three examinations, two or three leukocytes once, occasional red cells twice. There were no Bence-Jones bodies.

The blood examination showed 6,000 leukocytes, 85 per cent polynuclears, 13 per cent lymphocytes, 2 per cent mononuclears, 2,500,000 to 2,600,000 reds, hemoglobin 50 per cent, moderate achromia, very slight anisocytosis, platelets normal. A Hinton test was negative.

The temperature was 96° to 100° with a terminal rise to 104°. The pulse was 58 to a terminal 155. The respirations were normal except for a terminal rise to 38.

X-ray examination showed multiple areas of bone destruction in all the bones taken except the humerus. There were pathologic fractures of the sixth cervical, the fourth dorsal and the fifth lumbar vertebrae. Comparison with the previous films showed definite increase in the process.

The patient was placed on a Bradford frame with his neck immobilized. He was comfortable at first. No neurologic changes were detected. November 17 the pulse rose sharply. He complained of sharp pain on respiration along the right anterior axillary line. Nothing was heard in the chest. November 18 the temperature rose. November 19 portable x-rays showed the lung fields somewhat less radiant than at the time of the previous observations. The mediastinal shadow was increased in width and density. There was mottling extending outward from the lung roots. The diaphragm was high on the left and there was very little movement on that side. The entire left lung field was smaller than the right.

That evening the patient became very toxic and rather suddenly died.

Discussion

Dr. Chester M. Jones: I remember his general appearance when he came into the medical ward with a broken neck. I remember that moving his head very slightly caused intense pain, although he looked surprisingly well for his general condition. But beyond that I know nothing.

Dr. Edward G. Thorp: I talked with him a good deal. He told me that he did not fall and did not strike anything. He merely jerked his head sharply to one side to regain his balance.

Dr. George W. Holmes: The x-rays we have here cover a period of about a year and a half or a little over, and they show a picture which is rather characteristic of multiple medullary myeloma, but it may also be produced by metastatic carcinoma. He had no metastasis in the lung at that time. That was in March 1929, at the early stage of his illness. I think that at that time we were not at all sure the ribs were involved, though they look somewhat mottled. In the later stage it is quite obvious that the ribs are involved in the process and there is

also probably some involvement of the spine, although that is not at all certain. In his pelvis the picture is still more characteristic. There are obvious punched-out areas of diminished density scattered throughout the pelvis. This process of myeloma differs from the average case of carcinoma in that the lesions are more widely distributed. They are apt to be finer and show less reaction of the parts around them. He has here a rather unusual area in that it is quite large and begins to have more of a cystic appearance than the others.

All the films taken on the day of his injury, November 6, show quite extensive involvement of the vertebrae. There is no evidence of dislocation or displacement, but there are some mushroomed bodies with extensive involvement of the kind you would expect to cause flattening out of the bodies with slight trauma.

We have had some very interesting cases of myeloma lately in which the tumors looked like giant cell tumors and were single lesions, also another group in which there was lymphatic involvement and in which the pathologic diagnosis was myeloma. This latter group looks much like lymphoblastoma and responded very well to irradiation.

Dr. Channing C. Simmons: There are at present two other patients in the hospital with plasma cell myeloma, and these three cases illustrate well different forms of the disease. The case that has already been presented is the typical form of multiple myeloma. The other cases illustrate a myeloma of a single bone and the extra-osseous type.

I have been able to obtain some additional history on the case under discussion. He reported to the Outpatient Department in 1922 complaining of pain in his left shoulder and chest. A diagnosis of subdeltoid bursitis was made at this time. He next reported in 1929, at which time the correct diagnosis was made. The disease, therefore, is of at least eight years' duration.

The history of sudden pain in the neck following a jerk of the head is interesting. On questioning him, he said that any sudden movement of the head or shoulders would often be followed by acute pain in the neck or chest. These probably represented slight pathological fractures of the ribs following muscular exertion.

This is a typical case of multiple plasma cell myeloma, with the exception that no Bence-Jones bodies were found in the urine. Ewing states that Bence-Jones bodies are present in less than 50 per cent of the cases, but I personally have seen them in only one instance.

Plasma cell myeloma of a single bone is in my experience more common than the classical type. This is well illustrated by a patient in the hospital at present. A colored man entered the hospital in 1925. X-ray at that time showed a destructive tumor of the second lumbar vertebra. A diagnosis of giant cell tumor was made by x-ray, and radiation treatment given. The man was very uncoöperative and was not seen in the hospital again until August 1930. The vertebra at this time was collapsed. A stabilizing operation was performed and the tissues removed showed the tumor to be a plasma cell myeloma. Skeletal x-rays showed no other bone involvement.

There is a case on the Nerve Service at the present time that illustrates the extra-osseous type of the disease. This patient is a woman forty-seven years old who presented all the symptoms of a cord tumor of three months' duration. At operation an intraspinal tumor was found not attached to the bone which proved to be a plasma cell myeloma. Skeletal x-rays were negative.

I have here the x-rays of a patient admitted to the hospital in 1927. The entire humerus is destroyed by tumor, but all the other bones are normal. Shoulder joint amputation was performed to remove a sloughing, painful tumor. Microscopic examination showed plasma cell myeloma. The patient died one year later. The x-rays show bone destruction with no new bone formation. The head of the humerus shows a hemorrhagic soft mushy tumor extending from the head down to the elbow joint.

There is another case of considerable interest that was in the hospital some years ago, that of a man who in 1914-15 was operated upon several times for plasma cell myeloma in the skull and lower jaw. He is well today. Complete skeletal x-rays taken in 1928 showed no evidence of disease.

In other words, I believe that plasma cell tumor of the bone and soft parts is not altogether understood at the present time.

Dr. Tracy B. Mallory: That is certainly true. For a long time we thought plasma cell myeloma was a definite single entity and that it was always primary in and usually restricted to the bones. Then we began to pick up occasional biopsies on the lymph nodes of cases apparently clinically typical of lymphoma where the tumors were composed entirely of plasma cells that looked exactly like multiple myeloma of bone. Rather recently a case was autopsied at another Boston hospital where a diagnosis of plasmocytoma was made on the tonsils eight years before death. The patient died with perfectly typical multiple myeloma involving the sternum, ribs, and other bones.

Our case of today showed a typical plasma cell myeloma with extensive rib and vertebral involvement complicated by spontaneous fractures. He had a renal lesion, as is supposed to be characteristic of the disease, but it was a chronic glomerular nephritis rather than a nephrosis such as has been reported in several recent cases. There was a slight terminal bronchopneumonia.

Dr. Holmes: In the case which had a diagnosis of giant tumor of the spine the patient was

asked to wear a cast for the purpose of supporting his spine. We realized that he was going to have mushrooming of the vertebrae. He was fitted for a brace, but he would not wear it. We thought at the time that he did respond somewhat to radiation treatment. It may be that these tumors, like so many others, when they involve the bone do not respond well to radiation.

DR. SIMMONS: These were moderately undifferentiated cells and should respond to radiation. Ewing's sarcoma does.

DR. HOLMES: That is an exception. It could not be that those groups are different stages of the same disease?

DR. MALLORY: I do not know. I think it is possible. We have no real proof that this disease is always primary involvement of the bone. It might conceivably be primary in a lymph node but metastasize to the bones so early and extensively that the bony lesions predominate in the picture.

DR. SIMMONS: Doesn't Ewing describe a small round cell form of myeloma as well as the plasma cell type?

DR. MALLORY: Yes, but he uses myeloma in the sense of any tumor primary in the bone marrow, including myeloblastoma, lymphoblastoma and things of that sort as well. We have had two tumors of the spine recently, one in a child, the other that one Dr. Simmons spoke of in a middle-aged person. By x-ray and even surgical exploration there was no evidence of bone involvement, the major part of the growth being in the spinal canal. Both of them came to autopsy. In each case the marrow was extensively infiltrated with tumor but there was practically no bone destruction, so that the nature of the process would not have shown much by x-ray. Both were suggestive of myeloma, but neither was quite typical. They might well have been poorly differentiated myeloblastomas.

DR. SIMMONS: There is another case of cord tumor that came to the Nerve Department and was operated upon in which multiple areas of bone destruction were found but no tissue could be removed for diagnosis.

DR. MALLORY: Plasma cell myelomas have a characteristic age incidence in that they are quite rare below forty, fifty being about the average age. We have had one case in the past few months of a child of thirteen with some sort of lesion of the knee from whom an inguinal lymph node was removed. This showed a typical plasmocytoma, which is quite unusual at that age.

DR. SIMMONS: Dr. Mallory, in these two cases the patients' ages were reported as forty-two and forty-six when the symptoms began.

DR. MALLORY: They began a little earlier than usual.

DR. JONES: Have you any idea how often Bence-Jones bodies are found in such cases? We test for them frequently, but I cannot recall very many positive findings.

DR. MCKITTRICK: Ewing says they occur in less than 50 per cent of the cases.

DR. HOLMES: I remember that in one of the first cases we had in this hospital we found Bence-Jones bodies.

DR. SIMMONS: I should like to ask the Medical Department if there are any characteristic blood changes?

DR. BOCK: Sometimes we find suggestions of plasma cells.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Multiple myeloma.
Pathologic fracture of cervical vertebrae.
Bronchopneumonia.

ANATOMIC DIAGNOSES

Multiple plasma cell myeloma.
Chronic glomerulonephritis.
Hypertrophy of the heart, slight.
Bronchopneumonia.

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MEDICINE AND WAR

IN some technological processes it has been found that the by-product is of more value than the product for which the process was devised. So in certain non-material processes, as in education, it is sometimes the by-product, as we say, that is valued more highly than the learning itself. This is exemplified by the definition of education which indicates that "it is what is left after you have forgotten all you have learned". These "by-products" may be connected with the "side shows" of education.

There is, however, some confusion of thought and some invidious comparison implied by the terminology "by-product". Improvement in thinking ought to lead to improvement in nomenclature and it is more helpful to distinguish between the direct and the indirect methods for accomplishing results, in education as well as in other non-material processes.

An example that attracts attention now is that of a great foundation with medical interests. The field of its activities is widespread as indicated by the series of studies recently published[1], but can be seen more clearly in its annual report, namely as applied to a considerable part of the civilized and some of the uncivilized world. It promotes medical education and medical research, in all three lines of activity, preventive, curative, palliative, assisting in the dissemination of information as well as in the advance of knowledge, and including practical application and theoretical vision within its purview. Its contributions in the field of public health in providing resources for the carrying on of schools of public health, and in promoting interchange of ideas and personnel all over the world are well known.

What are some of the indirect results of this use of power which money gives? It is alleged that the power of certain foundations has been put to sinister uses[2]. "Sinister" has unjustifiable implications. Perhaps some of the results were "unexpected and undesirable", rather than "sinister".

One possible and important indirect effect lies in the prevention of war by promoting this very interchange of ideas and personnel, leading to mutual and reciprocal understanding and good-will and coöperation. This indeed is to take a long-time view, but it is the spirit of medicine, of pity for the weak and helpless, of consideration for the unfortunate and handicapped, of desire to bind up their wounds (of whatever nature), of cheerful courage and quiet determination to relieve and cure and prevent, that the world needs most today. Of such spirit the man of medicine has no monopoly, but the wisdom of the strategy of the Foundation lies in this: in promoting human physical well-being over all the earth, there is laid the surest foundation for the "well-being of mankind throughout the world", which is the expressed goal of its endeavors.

REFERENCES

1 Methods and Problems of Medical Education. (Eighteenth Series.)
2 Book Review—J. B. Clark. "Our New Progress." (New Eng. J. Med. 203:1318, (Dec. 25) 1930.)

IMPORTANCE OF CARE DURING PREGNANCY

IN the December issue of the *American Journal of Obstetrics and Gynecology* is an important article by Dublin and Corbin presenting a study, made jointly by the Maternity Centre Association of New York City and by the Statistical Bureau of the Metropolitan Life Insurance Company, of the records of 4726 women cared for by the Association from January 1, 1922 to December 31, 1929. Of these patients, about an equal number were delivered in their homes and in hospitals. About three-quarters of the total were attended by physicians and the remainder by midwives. During this period, no patient under care died before delivery. There were 11 maternal deaths after delivery of which 4 were due to sepsis, two to hemorrhage, two to embolism, and two to pulmonary causes. No

patient in the series developed eclampsia although a large number were treated for toxic symptoms. Of 4596 babies born alive, 132 died under one month of age. There were 123 stillbirths, of which the majority were, apparently, due to mechanical difficulties at delivery.

This series of cases is compared with a group of mothers in the same district who did not receive the care of the Maternity Association. During the period of comparison, the maternal mortality was nearly three times as high as the rate for the group of mothers receiving pregnancy care.

These data emphasize the incalculable importance of adequate supervision during pregnancy as a means of reducing maternal morbidity and mortality, and undoubtedly also the infantile mortality as well. It is our belief that the Pregnancy Clinic established by the Boston Lying-In Hospital in 1910, and maintained uninterruptedly ever since, was the pioneer demonstration of the value of this type of work in the United States, and perhaps in the world. Inevitably, however, only a small proportion of the total number of maternity cases can or should come under institutional care. The ultimate importance of such activities is the lesson which they should teach to private physicians throughout the country as to their own duties in the supervision and advice of patients who may be or who may become pregnant. Hitherto in pregnancy care, so far as it has been carried out, attention has been concentrated chiefly on toxemia, antepartum bleeding, and pelvic disproportions. In addition to these obvious points of pregnancy care, both in clinics and in private practice, attention should also be directed to dietetic advice as a means of limiting foetal weight and thereby reducing mechanical mortality at birth; and to the detection and elimination of maternal, focal infection, which may be not only a cause of general disabilities, but which may become a source of metastatic puerperal sepsis. As in all matters of preventive medicine, the fundamental and ultimate duty to the Community rests in the last instance on the shoulders of the private practitioner.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

SULLIVAN, ALBERT J. B.S., M.D. Harvard University School of Medicine 1927. Associate Physician, New Haven Hospital and New Haven Dispensary. Instructor in Medicine, Yale University School of Medicine. His subject is: "The Rôle of Surgery in the Treatment of Peptic Ulcer." Page 191. Address: New Haven Hospital, New Haven, Conn.

REID, WILLIAM D. A.B., M.D. Harvard University School of Medicine 1909. F.A.C.P. Assistant Professor of Cardiology, Boston University School of Medicine. Visiting Physician, Massachusetts Memorial Hospitals. Associate Member, Evans Memorial Hospital. Cardiac Consultant to the Newton Hospital and the Waltham Hospital. His subject is: "The Differential Diagnosis of Subacute Bacterial Heart Disease and Banti's Disease. Case Report." Page 198. Address: 510 Commonwealth Avenue, Boston.

FINKEL, HENRY S. M.D. Harvard University School of Medicine 1920. Gynecologist to Out-Patient Department, Beth Israel Hospital. Visiting Obstetrician, Evangeline Booth Hospital. Instructor in Obstetrics, Tufts College Medical School. His subject is: "The Diagnosis of Pregnancy by the Aschheim-Zondek Test." Page 203. Address: 21 Bay State Road, Boston.

BICELOW, GEORGE H. A.B., M.D. Harvard University School of Medicine 1916. Doctor of Public Health, Harvard 1921. Commissioner of Public Health for Massachusetts. Address: State House, Boston. Associated with him is:

HAMBLEN, ANGELINE D. A.B., Radcliffe, 1915. Member of Statistical Clinic, Massachusetts Department of Public Health, 1920. Address: Room 546, State House, Boston. Their subject is: "Our Falling Birth Rate." Page 209.

HOLMES, ARTHUR D. B.S., Ph.D. Laboratory Assistant, Dartmouth College, 1905-1906. Instructor in Chemistry, University of Maine 1906-1907. Instructor in Chemistry, Massachusetts Agricultural College 1907-1908. Assistant in Chemistry, Johns Hopkins University 1909-1911. Research Chemist, U. S. Department of Agriculture 1911-1918; E. I. duPont Company 1918-1921. Now Director of Research, The E. L. Patch Company. His subject is: "Are Vitamin D and Irradiated Ergosterol Identical?" Page 211. Address: The E. L. Patch Company, Stoneham, Mass.

BACON, THEODORE S. S.B., M.D. Harvard University School of Medicine 1898. F.A.C.P. Visiting Physician, Springfield Hospital. Address: 69 Maple Street, Springfield, Mass. Associated with him is:

CORLISS, PHILIP GORDON. M.D. Harvard University School of Medicine 1930. House Officer, Springfield Hospital 1930-1931. Address: 759 Chestnut Street, Springfield, Mass. Their subject is: "Perforation of the Intestine By a Fishbone." Page 214.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A STATED meeting of the Council will be held in John Ware Hall, Boston Medical Library, Wednesday, February 4, 1931, at 12 o'clock, noon.

Business:

1. Reading record of last meeting in abstract.

2. Report of Committee of Arrangements for the Sesquicentennial next June.
3. Report of Committee on Membership and Finance, on Membership.
4. Petitions for restoration to the privileges of fellowship.
5. Appointment of three delegates and three alternates to the House of Delegates, American Medical Association, for two years from June 1, 1931.
6. Appointment of delegates to the annual meetings of the New England State Medical Societies.
7. Appointment of delegates to the Annual Congress on Medical Education, Medical Licensure and Hospitals of the American Medical Association, at Chicago in February, 1931.
8. Report of the Treasurer and of the Auditing Committee.
9. Report of the Committee on Membership and Finance, on Finance.
10. Report of Committee on Clinics, Health Associations, and Industrial Clinics.
11. Report of Committee to examine, sort, and arrange papers and possessions in the vault at the Medical Library.
12. Report of the Committee on Public Education of the standing Committee on Public Health.
13. *Incidental Business.*

WALTER L. BURRAGE, *Secretary.*

Brookline, January 28, 1931.

A TOAST TO THE MASSACHUSETTS MEDICAL SOCIETY BY OLIVER WENDELL HOLMES

THE following letter from Dr. Holmes has come to light in the arranging of the papers in the vault at the Boston Medical Library:

296 Beacon Street, June 5th, 1883.
Francis H. Brown, M.D.
Anniversary Chairman etc.

Dear Sir:

I cannot be present I regret to say, at this year's meeting of the Massachusetts Medical Society.

If it is still the fashion to give what used to be called "Toasts" or "Sentiments," I will offer this:

The Massachusetts Medical Society,—May its members always and all of them bear in mind that Medicine is a Science as well as an Art and a means of getting a livelihood, and that all its alliances should be made with due respect to the stern and uncompromising demands of the august Sisterhood of the true Sciences, as well as to the convenience or kindly instincts of the practitioner.

Very truly yours,
O. W. HOLMES.

THE BOSTON MEDICAL LIBRARY

CLAUDIUS GALEN
131-200 A.D.

IT is 1800 years since Claudius Galen first drew breath which fact in itself is worth recalling in these days of tercentenaries and sesquicentennials. Few men have made an impression on their own times, not to mention those of 1400-1500 years thereafter, to be compared with that which Galen made. Certainly this is true of the members of the medical profession. Hippocrates, whom he sought to interpret and who had preceded him only 530 years or thereabouts, is the only one who can rival him in this respect. There were numerous factors which contributed to this prolonged interest in the life and activities of one man. In the first place he was known among his intimates as a philosopher, and one of no mean attainments or he could scarce have been so great a friend, as he appears to have been, to the Emperor Marcus Aurelius. He doubtless shone with a reflected glory as well as from the brilliancy of his own achievements. He was a voluminous and polished author, able to communicate his thoughts in equally good Greek or Latin and finally he practiced at a time in Rome when there were no physicians who could by any stretch of the imagination be compared with him. They hated him, made it as uncomfortable as they could for him and in derision characterized his successes in practice as "Galenic divinations", attributing them to dreams and occult influences of one sort and another. In one of his writings which is a close analogue of a portion of the 6th book of Plato's Republic, he depicts a philosopher-doctor, stranded in Rome (who is of course himself) whom he compares with Plato's philosopher, both likened to men seeking shelter from a storm beneath a wall. The storms from which Galen sought shelter were generally those of his own raising, for he abused the "profession" then practicing in Rome most shamefully. He criticized their intelligence and education, saying that many of them had taken to medicine before they could read or write and he attributed their inability to predict the days of crisis in the course of the fevers peculiar to Rome to their inability to count. Such statements were not calculated to increase his popularity with confreres and it is perhaps not to be wondered at that he was anxious to get back to his native town where friction of this type, at any rate, would not exist. His writings were numerous and he is perhaps the only author who has written a book about his own books. This was made necessary from the fact that as there was nothing to hinder plagiarism, it was freely practiced. He resorted to this measure in order to identify his own writings in his own time and for posterity.

When he came to Rome he left some of his manuscripts in Pergamos. Of these certain ones

were stolen by a servant and he lost others in a fire that destroyed the temple of Vesta, during the reign of Commodus. The existence of discrepancies in his writings he freely admitted and explained them on the ground that as his experience increased and his judgments became more mature he could not adhere to his earlier beliefs. He relates in his books a great deal that is of interest as to the history and practice of medicine in the Greek and Graeco-Roman world, how it came to be what it was in his day, what methods of research were practiced, who were the favored teachers, what the outstanding topics of discussion were in philosophy and medicine, who were numbered among the Roman Savants and what were their manners and resorts, with frequent mention of the petty jealousies and medical rivalries current in Rome at that period. There were many sects in his time for which he had no use and with none of which he would be identified and one of his outstanding characteristics, namely his independence, was indicated in an aphorism of his own, viz.: "Call no man master." He was keenly interested in anatomy and thought it valuable from four standpoints, first, from the purely anatomical standpoint; second, from the standpoint of biology; third, from the practical, surgical standpoint and lastly from that of the philosopher. He himself made some contributions to practical, descriptive anatomy and relates how he, on two occasions, was fortunate enough to secure for study, bodies that had met a violent death and were not discovered until they had been divested of most of the flesh by animals and the elements. He was thus enabled to secure two skeletons without however any opportunity for dissection of the soft parts. There were only three manuals on anatomy in existence in Galen's day, the first and most important was by Marinus. The views of the authors of these were so much at variance that a part, at least, of Galen's work in anatomy was the systematization of these manuals. He urged his students to go to Alexandria where were to be found better opportunities for the study of human anatomy than elsewhere. Also he suggested that they follow the armies in the field. From the suggestions he makes as to the best variety of ape to select for purposes of dissection it is evident that he neglected no opportunity to study comparative anatomy. He killed the apes he employed in this way by drowning in order to avoid injury to structures which he desired to observe. At the gladiatorial contests he was a frequent and privileged attendant for he learned that it was often possible in the treatment of their wounds to make anatomical observations that might be instructive. Galen's investigative spirit led him into the physiological field and though many of his theories are fantastical enough, yet, on the whole, he deserves to be praised for what he knew rather than censured for what he failed to understand. For example he had a good idea of, and described fairly well, the type of digestion that goes on in the stomach and it is scarcely likely that he could have gone any further than he did without the aid of the microscope. He had, as did many of the "ancients", a better understanding of the circulation than has been credited to them. The step which in its complete conception was not taken definitely until Harvey's time, was the one that cleared up the pulmonary circulation. Just how near Galen came to an understanding of these facts is not known. The simplicity of his ideas, the independence of his position, the keenness of his powers of observation, his genius for work and his many and various contributions to medical literature secured for him a preëminence that undoubtedly kept back the world's progress in medicine for many, many years.

Galen was born in Pergamos in the year 131 A.D. His father, Nicon, was an able man. He was an architect, geometer and a savant. His mother seemed of an inferior type. He counted among his most conspicuous advantages the liberal culture which he acquired through his early training. He looked upon the tendency to specialization, which was even then beginning to manifest itself, as an early indication of the decadence of the Roman people. An early interest in philosophy determined the bent of his mind and Plato was destined to strongly influence his manner of thinking. He was brought up in the midst of numerous philosophical schools and was in the habit of hearing the Peripatetics, Stoics and Platonists disagree about most everything except mathematics, so he turned his attention to that subject, thinking if it was the *one* topic upon which they could all agree it must be worth while. He was 17 when he began the study of medicine and was as firmly convinced that he had a divine call to do so as Socrates was firm in his belief that he was possessed by a demon. He studied anatomy in Pergamos under Satyrus who was a pupil of the famous Quintus. From here he went to Corinth, Smyrna and finally to Alexandria where he finished his medical education. He came back to Pergamos and started his practice and it was here that he was appointed surgeon to the gladiators. He remained four years and then went to Rome in his 31st year. This was at the very beginning of the reign of Marcus Aurelius. During the next five years he rose to a position of importance, in large part due to the influence of one of the principal literary circles of the Capital. Roman culture which had commenced to wane received a temporary stimulus from the Emperor Aurelius. Galen's intimates in this literary circle were Flavius Boetthus, Alexander of Damascus, a professor of Philosophy at Athens, and Sergius Paullus, prefect of the City. They claimed he was a skilled physician because he was a born philosopher and brought his abilities to the attention of Marcus Aurelius. For

the first two years he was in Rome he gave public, anatomical demonstrations and practiced his profession. He gives in his book on Prognosis several glimpses of the "fashionable" practice which was so largely his in Rome, indicating that his reputation as a practitioner of the "black art" rested on his habit of close observation when in the sick room. He seems to have been particularly keen in the matter of prognosis and from his study of fevers was able to foretell, with what appeared to his less skilled contemporaries, an almost uncanny accuracy, the exact day when a given fever would reach its crisis. His introduction to the Philosopher-Emperor, Marcus Aurelius, was occasioned by the unphilosophic ingestion of cheese which induced a stomach ache. His bedside manner must have been pleasing for the Emperor in dismissing him remarked, "I have only one physician and he is a gentleman." The life in Rome apparently could not hold Galen's interest for long and he frequently spoke to his friends of his determination to return to the quieter life of his native city. When he was 37, in the year 168 A.D., he secretly made his escape, first to Campania, then to Cassiope in Corcyra and at last to Pergamos. He was not allowed to remain, for an Imperial summons recalled him to Rome a year later. It was the Emperor's intention to take Galen with him on an expedition to Germany but the death of Verus called Aurelius back to Rome and Galen came with him. When Aurelius set out a second time for Germany he yielded to Galen's importunity and left him behind in charge of the youthful Commodus. He entered at this time upon a period of great literary activity and was away from the capital as much as he could be. From this time there is but little by which to trace his career. Whether he died in Rome or exactly how long or where he lived is very uncertain. There are some indications that he traveled more or less and it is certain that he made at least one other visit to his native place after his return to Rome.

It is a difficult matter to appraise the accomplishments of a man like Galen, if comparisons are to be made with modern discoveries in medical science. It should not be the amount of knowledge that one possessed as compared with what a modern knows, but comparisons should be based on what a given person discovered that was sound. Viewed in this way the "ancients" compare very favorably with any in these days. Galen's interest in anatomy was a paramount one and dissection was a passion with him. His familiarity with it contributed very considerably to his admiration for the functional adaptation of certain anatomical structures with which he had acquired special familiarity. In physiology however he perhaps contributed more to the source of human knowledge than in any other field and he was enabled to do this because in addition to being an accurate observer he was possessed by a desire to investigate in order that he might find an explanation of the phenomena that were so constantly recurring in his practice. His observations upon the heart and its action, the occasional patency of the foramen ovale with the explanation he offers for it, the anastomosis of veins and arteries, numerous citations which show that arteries and veins were well known long before his own time, the proof that air, or "spiritus", was not the activating substance that the arteries contained (that in fact they did not contain air at all), that ligature of vessels was common practice in his time in order to check haemorrhage, to be forgotten until revived by Paré, and many more equally vital physiological observations upon the anatomy of the nervous system, prove that Galen as a research scholar would have made his mark in any age. It naturally follows that he was an outspoken opponent of quackery in all of its forms.

From the time of his death, which was in all probability about the end of the second century A.D., until the beginning of the revival of learning, particularly until the time when Vesalius began to cast doubt upon his work as a whole, he was unquestionably the undisputed authority in medicine. By discrediting many of Galen's anatomical observations, which might readily be done because all his work was accomplished before dissection of the human body had been undertaken, it was not difficult to raise a question as to how accurate his other observations might have been, without disparaging the discoveries of Harvey or Vesalius it certainly is not unfair to give Galen credit for having himself more nearly fulfilled the rôle of a genius than did any of those who during the period, from 1200 to 1400, years after his time, were lauded for having dislodged him from his position of preëminence.

MASSACHUSETTS LEGISLATIVE NOTES

HOUSE—NO. 914

The *Journal* is disappointed in not being able to present the text of the Chiropractic bill in this issue for the reason that a redraft was necessary and the final draft is not yet available.

HOUSE—NO. 576

By Mr. Connolly of Boston, petition of John P. Connolly for legislation relative to the time within which decisions shall be rendered by the Industrial Accident Board. (Labor and Industries.)

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Thirty-One.

An Act relative to the Time within which Opinions shall be rendered by the Industrial Accident Board.

Be it enacted by the Senate and House of Repre-

sentatives in General Court assembled, and by the authority of the same, as follows:

Section ten of chapter one hundred and fifty-two of the General Laws, as amended by chapter two hundred and eight of the acts of nineteen hundred and thirty, is hereby further amended by inserting in the sixth line after the word "decision" the words:—within three weeks,—so as to read as follows:—Section 10. If a claim for a review is filed under section eight, the reviewing board shall hear the parties, and may hear evidence in regard to pertinent matters and may revise the decision in whole or in part, or may refer the matter back to the member for further findings of fact, and shall file its decision within three weeks with the records of the proceedings and notify the parties. If a claim for a review is so filed by the insurer in any case and the board by its decision orders the insurer to make, or to continue, payments to the injured employee, the cost to the injured employee of such review, including therein reasonable counsel fees, shall be determined by the board and shall be paid by the insurer. No party shall as of right be entitled to a second hearing upon questions of fact.

House 450 provides for additional members of the Industrial Accident Board which shall consist of nine members, one of whom shall be a woman.

House 476 provides for an amendment of Section 1 of Chapter one hundred and eleven of the General Laws and would give to the county commissioners additional power to provide diagnostic service with respect to tuberculosis in coöperation with the local boards of health especially with reference to the establishment and maintenance of out-patient departments.

HOUSE NO. 494

By Mr. Holden of Attleboro, petition of Henry M. Pollock and others for legislation to provide liens in favor of hospitals for the treatment and maintenance of persons injured in accidents. Judiciary (Joint).

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Thirty-One

An Act to provide for Liens in Favor of Hospitals for Treatment and Maintenance of Persons injured in Accidents.

Be it enacted by the Senate and House of Representatives in General Court assembled, and by the authority of the same, as follows:

Section 1. Every charitable association, corporation or other institution maintaining a hospital in the commonwealth of Massachusetts supported in whole or in part by private charity or public funds shall be entitled to a lien upon any and all rights of action, suits, claims and demands of every person admitted to any such hospital and receiving treatment, care and maintenance therein on account of any personal injuries received in any accident against any other person or corporation which any such injured person may or shall have, assert or maintain against any such other person, or corporation for damages on account of such injuries, for the amount of the charges of such hospital for such treatment, care and maintenance, provided that notice in writing of a claim of lien containing the name and address of the injured person, date of the accident, name and location of the hospital, and, if known, the name of the person or persons, firm or firms against whom, and the corporation or corporations against which, such action, suit, claim or demand is made, shall be filed in the office of the clerk of the superior court of the county in which such injury shall have occurred, prior to the payment of any moneys to such injured person or his or her legal representative therefor.

After the filing of such notice it shall be the duty of the hospital to mail, postage prepaid, a copy of such notice with a statement of the date of the filing thereof to the person or persons, firm or firms, corporation or corporations, alleged to be liable for the injury sustained by such injured person if such names and addresses shall be known.

Section 2. The lien of any such hospital shall attach to all property paid, transferred or delivered in settlement of any such claim or demand effected by or in behalf of any such injured person or by his executors, administrators or assigns; and also shall attach to any verdict, report, decision, decree, award, judgment, or final order made or rendered in any action or proceeding in any court of record in Massachusetts or any public board or bureau in any suit, action, or proceeding brought by such injured person or by the estate of such injured person, and no execution shall issue thereon until such lien shall have been released.

Section 3. After the filing of the notice as herein provided no release of any claim or demand on account of such injuries or in respect of any such verdict of a court, decision, decree, award, judgment or final order made or rendered as hereinbefore mentioned executed by or on behalf of any such injured person or by his or her estate shall be valid or effectual between the parties thereto or otherwise unless prior to the execution and delivery thereof all such charges of any such hospital or institution shall have been paid in full or the lien of such hospital shall have been released; and the payment transfer or delivery of any property subject to this lien to any person or corporations other than the hospital entitled to such lien shall render the person or corporation making the same liable to pay to the hospital the full amount of such hospital charges.

Section 4. The clerk of the superior court in each county of the commonwealth shall provide a suitable book, to be called the Hospital Lien Docket, in which shall be entered the name of the injured person, the date of the accident, the name of the hospital making the claim and the amount thereof immediately upon filing of papers hereunder stating the same.

Section 5. A lien under this act shall become void unless within thirty days after the injured person has been discharged from the hospital making the claim or within thirty days after termination of hos-

pital treatment the hospital shall file with the clerk of court in which the claim shall have been filed a statement of the amount of the lien so claimed, but otherwise it shall remain in full force and virtue until settlement of the same has been paid.

Section 6. This act shall not apply to accident cases coming within the provisions of acts relating to workmen's compensation.

Section 7. This act shall take effect upon its passage.

MISCELLANY

INFLUENZA

Following a year marked by a gratifying reduction of morbidity and mortality January is showing a somewhat widespread incidence of influenza. Up to the time of this writing the United States is not suffering to the extent of epidemic conditions in Europe. Thus far the disease has not shown the malignancy of that of 1918.

MASSACHUSETTS GENERAL HOSPITAL TRAINING SCHOOL FOR NURSES

Graduation exercises of the training school for nurses of the Massachusetts General Hospital will take place Friday evening, February 6, 1931, in the Moseley Memorial Building, Fruit Street, Boston.

WHITE HOUSE CONFERENCE ON CHILD HEALTH AND PROTECTION

PRELIMINARY PROGRAM OF FINAL SESSION

SECTION I—MEDICAL SERVICE

February 19, 20, 21, 1931
Washington, D. C.

Samuel McC. Hamill, M.D., Chairman

Committee A—Growth and Development
Kenneth D. Blackfan, M.D., Chairman, Professor of Pediatrics, Harvard University Medical School.

Committee B—Prenatal and Maternal Care
Fred Lyman Adair, M.D., Chairman, Professor of Obstetrics and Gynecology, the University of Chicago.

Committee C—Medical Care for Children
Philip Van Ingen, M.D., Chairman, Clinical Professor, Diseases of Children, Columbia University College of Physicians and Surgeons.

Thursday, February 19, 1931

9:00 A. M. Opening Session for All Committees—Presiding, Samuel McC. Hamill, M.D., Chairman of Section on Medical Service.

Address of Welcome—Ray Lyman Wilbur, M.D., Chairman of the Conference.

COMMITTEE A—GROWTH AND DEVELOPMENT

Thursday, February 19, 1931

9:30—11:00 A. M. Presiding, Henry F. Helmholz, M.D., Professor of Pediatrics, University of Minnesota Graduate School of Medicine.

General Considerations — Edwin Bidwell Wilson, Ph.D., Professor of Vital Statistics, Harvard School of Public Health.

Definition of terms, use of statistical procedure, heredity, the well-born child, the significance of different age levels, the influence of climatic factors and socio-economic circumstances.

Discussion.

11:00 A. M.—1:00 P. M.

Anatomical Considerations—Richard E. Scammon, Ph.D., Professor of Anatomy, the University of Chicago.

The morphological phases of growth which have a direct bearing upon child health and protection. The growth of individual organ systems of the body. Anatomical considerations in respect to human types, body mechanics, differential growth and prematurity. The more common pathological processes which modify growth. The sequence of events under normal circumstances.

Discussion.

2:30—4:30 P. M. Presiding, Walter Bradford Cannon, M.D., ScD., Professor of Physiology, Harvard University Medical School.

Physiological Considerations—A. J. Carlson, M.D., Sc.D., Professor of Physiology, the University of Chicago.

Development of the physiological processes under normal conditions as well as in the presence of the more common pathological disturbances. The physiology of each of the systems of the body. Physiological adjustment, temperature regulation, fatigue, sleep, and prematurity as they directly bear upon the practical problem of child health.

Discussion.

8:00 P. M. (See Program of Committee C—Medical Care for Children.)

Friday, February 20, 1931

(See Program of Committee B—Prenatal and Maternal Care.)

9:30 A. M. (See Program of Committee C—Medical Care for Children.)

2:00—3:30 P. M. Presiding, Frank N. Freeman, Ph.D., Professor of Educational Psychology, the University of Chicago.

Appraisal of the Child

1. Physical Status—T. Wingate Todd, M.B., F.R.C.S., Professor of Anatomy, Western Reserve University School of Medicine.

The standards by which physical status is to be judged, and various other measurements of value not acceptable as standards, are to be considered critically. Appraisal of the individual child with special reference to methods of examination and particular findings of significance at different ages. Application to groups of children.

Discussion.

3:30—5:00 P. M.

2. Mental Status—Douglas A. Thom, M.D., Director, Division for Mental Hygiene, Massachusetts Department of Mental Diseases.

The development of intelligence and its measurement by tests of various types. The development of motor skills and language. The part played by emotion, by habit; and by wise training in determining the behavior of children. Other subjects relating to mental development.

Discussion.

8:00 P. M. Presiding, John R. Murlin, Ph.D., Sc.D., Professor of Physiology, University of Rochester.

Nutrition—Lafayette B. Mendel, Ph.D., Sc.D., Professor of Physiological Chemistry, Yale University.

Certain general considerations as to sources and supply of food substances as well as the individual food constituents, reviewed both from the standpoint of our knowledge of the nature of these substances and their practical significance in the diet of children.

Discussion.

COMMITTEE B—PRENATAL AND MATERNAL CARE

Thursday, February 19, 1931

9:30—11:30 A. M. Presiding, Fred Lyman Adair, M.D., Chairman of the Committee.

Prenatal, Maternal and Early Infant Care—John Osborn Polak, M.D., Prof. of Obstetrics and Gynecology, Long Island College Hospital School of Medicine.

Consideration of methods for consecutive, prenatal, intranatal and postnatal care for institutional and home cases of different racial groups in urban and rural communities.

Discussion to be opened by: Adelaide Brown, M.D., Edward C. Lyon, Jr., M.D.

11:30 A. M.—12:30 P. M.

Interested Organization—Robert D. Mussey, M.D., Professor of Obstetrics, University of Minnesota (Mayo Foundation).

Presentation of accomplishments and programs of various organizations of national scope having activities related to prenatal and maternal care.

Discussion to be opened by: T. F. Murphy, M.D., Clara E. Hayes, M.D.

2:00—5:30 P. M. Presiding, Fred Lyman Adair, M.D., Chairman of the Committee.

Factors and Causes of Fetal, Early Infant and Maternal Morbidity and Mortality—Hugo Ehrenfest, M.D., Assistant Professor of Obstetrics and Gynecology, Washington University Medical School.

Consideration of antenatal causes and factors, intranatal causes and factors, postnatal causes and factors, morbidity and mortality statistics.

Discussion to be opened by: Fred J. Taussig, M.D., E. D. Plass, M.D., Frank W. Lynch, M.D.

8:00 P. M. (See Program of Committee C—Medical Care for Children.)

Friday, February 20, 1931

9:00—10:15 A. M. Presiding, Fred Lyman Adair, M.D., Chairman of the Committee.

Obstetric Teaching and Education of Midwives—James Robert McCord, M.D., Professor of Obstetrics and Gynecology, Emory University.

So long as midwives are necessary in some sections of the country and to care for certain racial groups, they must be trained in obstetric technic and work under medical supervision and control.

Discussion to be opened by: Ralph W. Lobenstine, M.D., J. P. Bowdoin, M.D.

10:15—11:30 A. M.

Obstetric Teaching and Education of Nurses and Nursing Attendants—George W. Kosmak, M.D., Editor, American Journal of Obstetrics and Gynecology.

Report and recommendations relative to the teaching and education necessary to insure adequate nursing service to mothers and infants in homes and institutions by those engaged in this profession as institutional, public health, private duty nurses or in other fields of nursing activities.

Discussion to be opened by: Hazel Corbin, R.N., Calvina MacDonald, R.N.

11:30 A. M.—12:30 P. M.

Teaching and Education of the Laity and Social Workers—Robert L. DeNormandie, M.D., Instructor in Obstetrics, Harvard University Medical School.

Report and recommendations regarding the subject matter and methods of instructing the laity and the stating of what and how and by whom the social workers should be taught about the problems which confront them in dealing with mothers and newly born infants.

Discussion to be opened by: Miss Kate McMahon, M. Pierce Rucker, M.D.

9:00 A. M.—12:30 P. M. Presiding, Leslie B. Arey, Ph.D., Professor of Anatomy, Northwestern University Medical School.

Basic Sciences and Their Relation to Maternal and Fetal Problems—Leslie B. Arey, Ph.D.

Report and recommendations of the committee relative to the part played by the basic sciences in the development of a program for child health and protection with suggestions for proper fields of research requiring stimulation and support.

Discussion to be opened by: E. T. Bell, M.D., I. G. Macy, Ph.D.

2:00—5:30 P. M. Presiding, Fred Lyman Adair, M.D., Chairman of the Committee.

Obstetric Teaching and Education of Physicians.

1. Correlation of Instruction in the Basic Sciences with that in Obstetrics from:
   a. Viewpoint of Basic Sciences—Leslie B. Arey, Ph.D., Professor of Anatomy, Northwestern University Medical School.
   b. Viewpoint of Obstetrics—J. M. H. Rowland, M.D., Professor of Obstetrics, University of Maryland School of Medicine.
2. Undergraduate Education—Palmer Findley, M.D., Pres. Central Association of Obstetrics and Gynecology.
3. Graduate Education—Rudolph W. Holmes, M.D., Associate Professor of Obstetrics, Northwestern University Medical School.
4. Education and other Obstetric Requirements for Granting Degree of Doctor of Medicine, Licensing to Practice, and for Specialization—Walter T. Dannreuther, M.D., Professor of Gynecology, New York Post-Graduate Medical School.

Discussion to be opened by: Reuben Peterson, M.D., J. Whitridge Williams, M.D., Sc.D.

8:00 P. M. (See Program of Committee A—Growth and Development.)

COMMITTEE C—MEDICAL CARE FOR CHILDREN

Thursday, February 19, 1931

9:30 A. M.—1:00 P. M. Presiding, Harold K. Faber, M.D., Prof. of Pediatrics, Stanford University School of Medicine.

Convalescent Care—Adrian V. S. Lambert, M.D., Chairman, Executive Committee on Convalescence, Welfare Council of New York City.

Types of institutional buildings; classes of patients received; length of stay; professional staff (services and responsibilities); staff for care, recreation and education; facilities for meeting emergencies; methods of admitting and discharging; relations between homes and referring agencies. Conclusions and recommendations.

Foster Homes for Convalescence—Ida M. Cannon, R.N., Chief of Social Service, Massachusetts General Hospital.

Discussion to be opened by: E. H. L. Corwin, Ph.D.

9:30 A. M.—1:00 P. M. Presiding, Herbert B. Wilcox, M.D., Professor Diseases of Children, Columbia University College of Physicians and Surgeons.

Psychiatry and Psychology—Bronson Crothers, M.D., Asst. Prof. of Pediatrics, Harvard University Medical School, Neurologist to Children's Hospital, Boston.

Report limited to discussion of duties and opportunities open to practitioners of medicine; confusion of field due to conflict of different groups; natural strength of the position of the pediatrician; relative simplicity of the problem is rationally approached.

Discussion to be opened by: Esther Loring Richards, M.D., D.Sc., Borden S. Veeder, M.D.

9:30 A. M.—1:00 P. M. Presiding, Lloyd T. Brown, M.D., Instructor in Orthopedic Surgery, Harvard University Medical School.

Orthopedics and Body Mechanics—Robert Bayley Osgood, M.D., Professor of Orthopedic Surgery, Harvard University Medical School.

Incidence of poor body mechanics; relation to functional health; what is being taught; what ought to be done in education of physicians, physical therapists, and school teachers; possibility of universal training of school children in principles of good body mechanics and in method of correction of poor body mechanics; results to be expected. Recommendations.

Discussion to be opened by: John Berton Carnett, M.D., Fritz B. Talbot, M.D.

9:30 A. M.—1:00 P. M. Presiding, Edna L. Foley, R.N., D.Sc., Superintendent Visiting Nurse Association of Chicago.

Nursing—Stella Goostray, R.N., Secretary, National League of Nursing Education, Superintendent, School of Nursing, Children's Hospital, Boston.

The functions of the nurse in the care of children; preparation—personal qualifications and general education, preparation in general courses of nursing, postgraduate instruction in pediatric nursing, postgraduate course in public health nursing; adequacy of services rendered by nurses—as judged by physicians, public health nursing agencies in cities, small towns, and rural areas. Conclusions and recommendations.

Discussion.

2:30—5:00 P. M. Presiding, Isaac A. Abt, M.D., Professor of Pediatrics, Northwestern University Medical School.

Hospitals and Dispensaries—Clifford G. Grulee, M.D. (Lantern Slides), Clinical Professor of Pediatrics, Rush Medical College.

Distribution of hospitals and dispensaries accepting children; organization for and extent of medical care for children; relation to pediatric education; preventive activities; facilities for convalescent care, for medical social service; organization for dental care; for correction of poor body mechanics. Conclusions and recommendations.

Discussion to be opened by: Adrian V. S. Lambert, M.D., Ida M. Cannon, R.N., Percy R. Howe, D.D.S., D.Sc., Robert Bayley Osgood, M.D.

8:00 P. M. Presiding, Ray Lyman Wilbur, M.D., Chairman of the Conference.

Preventive Measures—Philip Van Ingen, M.D., Chairman of the Committee.

Attitude of physicians, hospitals and dispensaries, and health centers toward:— health examinations of children, vaccination against smallpox, immunization against diphtheria. Attitude of parents toward these procedures.

Report on a National Survey to Determine the Extent to which Preventive Medical and Dental Services Reach Preschool Children—(Lantern Slides) George T. Palmer, Dr. P.H., Director of Research, American Child Health Association.

1. In 158 cities based upon approximately 85,000 families interviewed. Relative standing of states in proportion of preschool children having had health examinations, dental health examinations, vaccination against smallpox and diphtheria immunization. Influence of economic status and age.

2. In rural areas in 42 states based upon approximately 25,000 families interviewed. Relative standing of states in proportion of preschool children reached. Variations at different age levels. Geographic differences in hospital and home delivery service and attendance at birth. Accessibility of medical, dental and hospital service.

Conclusions and recommendations—Samuel McC. Hamill, M.D., Chairman of the Section.

Discussion.

Friday, February 20, 1931

9:30 A. M.—12:30 P. M. Presiding, Philip Van Ingen, M.D., Chairman of the Committee.

Pediatric Education—(Lantern Slides) Borden S. Veeder, M.D., Professor of Clinical Pediatrics, Washington University School of Medicine.

Undergraduate instruction:—adequacy of pediatric education as estimated by practising physicians; facilities for and methods of teaching pediatrics; time devoted to pediatrics; instruction in certain subjects, such as, the newborn, physical and mental development, public health and social factors, preventive measures; conclusions and recommendations. Postgraduate instruction:—opportunities, extent and type; demand as shown by attendance, etc.; conclusions and recommendations.

Discussion to be opened by: Alfred Stengel, M.D.; Isaac A. Abt, M.D.; Adrian V. S. Lambert, M.D.; Robert Bayley Osgood, M.D.; Bronson Crothers, M.D.; Ida M. Cannon, R.N.

2:15-5:00 P. M. Presiding, Edith M. Baker, President, American Assn. of Hospital Social Workers.

Medical Social Service—Ida M. Cannon, R.N., Chief of Social Service, Massachusetts General Hospital.

Extent and distribution; functions of medical social service worker; integration of social and medical work; relation to public health nursing activities; adequacy of present personnel; number and training; training facilities; educational facilities. Conclusions and recommendations.

Discussion to be opened by: Richard M. Smith, M.D., Porter Lee.

2:15-5:00 P. M. Presiding, William DeKleine, M.D., Medical Asst. to Vice Chairman, American Red Cross.

Health Centers—J. H. Mason Knox, Jr., M.D., Chief, Bureau of Child Hygiene, State of Maryland Dept. of Health; and Mrs. Berthold Strauss, Vice-President, Community Health Center, Philadelphia.

Types of organization, county, city, public and private; clientele served; relation to other health agencies, hospitals, dispensaries; county health programs, gaps in the service. Conclusions and recommendations.

Relation of the Nutritionist to the Health Program—Lucy H. Gillett, M.A., Superintendent, Nutrition Bureau Assn. for Improving the Condition of the Poor.

Discussion to be opened by: Ira V. Hiscock, M.D.; Mary Riggs Noble, M.D.; Charles Francis Wilinsky, M.D.

2:15-5:00 P. M. Presiding, Leroy M. S. Miner, M.D., D.M.D., Professor of Oral Surgery, Harvard University Dental Department.

Dentistry and Oral Hygiene—Percy R. Howe, D.D.S., D.Sc.

Extent of dental caries in children (reliability of statistics); value of therapeutic work in prevention of caries; educational trends in corrective and preventive work; scientific basis of present theory and practise, part of the medical practitioner in prevention of dental caries. Conclusions and recommendations.

Discussion to be opened by: Guy S. Millberry, D.D.S.

8:00 P. M. (See Program of Committee A—Growth and Development.)

Saturday, February 21, 1931

9:30 A. M. Joint Session for All Committees—Presiding, Ray Lyman Wilbur, M.D., Chairman of the Conference.

Summaries of Findings and Recommendations of the Committees

1. Committee A—Growth and Development—Kenneth D. Blackfan, M.D., Chairman of the Committee.
2. Committee B—Prenatal and Maternal Care—Fred Lyman Adair, M.D., Chairman of the Committee.
3. Committee C—Medical Care for Children—Philip Van Ingen, M.D., Chairman of the Committee.

These summaries are to indicate so far as is possible the gaps that exist in present procedures and suggestions as to how they may be filled in order to bring about a well-rounded nation-wide child health program.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1929 AND SEVEN YEAR AVERAGE

MONTH ENDING JANUARY 10, 1931

Diseases	Wk. ending Dec. 20, 1930	Wk. ending Dec. 27, 1930	Wk. ending Jan. 3, 1931	Wk. ending Jan. 10, 1931	Average cases reported for week corresponding to Jan. 10 for past seven years	Wk. ending Dec. 21, 1929	Wk. ending Dec. 28, 1929	Wk. ending Jan. 4, 1930	Wk. ending Jan. 11, 1930
Anthrax	—	—	—	—	—	—	1	—	—
Cerebrospinal Men.	—	—	—	1	1	3	3	2	1
Chicken pox	82	68	78	200	136	126	150	113	204
Conjunctivitis Inf.	—	—	—	25	4	2	1	1	—
Diphtheria	14	11	9	17	41	21	23	12	24
Dysentery Bacillary	—	1	—	—	—	—	—	—	—
Encephalitis Epid.	—	—	—	1	—	—	—	—	—
German Measles	4	3	5	3	23	6	12	24	31
Influenza	2	3	2	10	448	9	3	6	12
Measles	77	118	168	271	207	4	19	64	62
Mumps	48	16	29	134	50	19	6	12	59
Paratyphoid Fever	—	—	—	—	—	1	1	—	—
Pneumonia (Broncho)*	18	18	25	26	43	13	21	23	20
Pneumonia (Lobar)	23	28	31	42	60	36	39	47	37
Poliomyelitis	—	—	—	—	—	1	1	—	—
Scarlet Fever	87	55	57	57	118	85	63	84	129
Septic Sore Throat	2	5	1	4	3	2	—	1	2
Smallpox	—	—	—	—	8	—	—	—	—
Trachoma	—	—	—	—	—	—	1	—	—
Trichinosis	—	1	—	—	—	1	—	—	—
Tuberculosis (Pul.)	25	22	25	29	30	32	21	30	25
Tuberculosis (O. F.)	2	—	2	8	3	1	3	1	3
Typhoid Fever	3	1	2	—	2	2	—	—	—
Undulant Fever†	1	2	1	—	—	—	—	1	—
Whooping Cough	29	64	37	76	75	64	24	33	82
Gonorrhea	22	15	17	20	35	29	14	35	19
Syphilis	32	17	43	35	30	33	27	33	43

*Made reportable January 1, 1925.
†Made reportable July 1, 1928.

Remarks: No cases of Asiatic cholera, glanders, plague and yellow fever during the past seven years.

REQUEST FOR MORE HOSPITAL BEDS FOR VETERANS

Representative Edith Nourse Rogers of Massachusetts has petitioned Congress for more hospital facilities for Massachusetts Veterans.

This subject has been studied by the House Veterans Committee with the expectation that the fifty-two million dollars which has been suggested will be materially reduced.

While a grateful country should be generous to the veterans in providing all necessary hospital facilities, conditions warrant careful examination of the needs of the World War soldiers.

MORTALITY RATES

Telegraphic returns from 81 cities with a total population of thirty-six million for the week ending January 10, indicate a mortality rate of 14.1 as against a rate of 12.8 for the corresponding week of last year. The highest rate (26.5) appears for Trenton, N. J., and the lowest (5.8) for South Bend, Ind. The highest infant mortality rate (14.9) appears for Somerville, Mass., and the lowest for Canton, Ohio, Utica, N. Y., and Yonkers, N. Y., which reported no infant mortality.

The annual rate for 81 cities is 13.7 for the two weeks of 1931, as against a rate of 13.1 for the corresponding weeks of 1930.

HEARING ON THE MATERNITY BILL

Special to the *Transcript:*

Washington, January 19—Massachusetts will be among the leaders in the drive that opens Tuesday, in hearings before the House Committee on Interstate Commerce, to prevent action this session on the so-called Maternity Act. Having already passed the Senate, House action is now necessary. The Sen-

tinels of the Republic are among the organizations opposing the measure, which has, of course, strong support. In the House A. Piatt Andrew of Massachusetts has long fought this legislation, and in the Senate David I. Walsh of Massachusetts has actively campaigned against it. Andrew repeatedly fought extension of the maternity bill, as being one of a group of objectionable Federal aid proposals.

"An examination of the statistics presented by the Children's Bureau as to the rate of infant mortality in the different States will disclose how thoroughly futile has been their activity," Andrew says. "Five States have refused to accept the 50-50 benefits authorized by this act—Kansas, Illinois, Connecticut, Maine and Massachusetts. The figures furnished by the Children's Bureau do not demonstrate any remarkable results from Federal aid. They show that the highest infant mortality occurs in States that are receiving Federal aid, and that the infant death rate is considerably below the average in States that have refused Federal aid. Statistics would seem to indicate that if you want to preserve the babies, you would do well to avoid Federal aid."

CITES LEGISLATURE'S ACTION

Senator Walsh spoke against the bill during debate in the Senate, and placed in the record statements in opposition from the Massachusetts Medical Society, the Massachusetts Civic Alliance and other organizations. He took occasion to point out that the Massachusetts Legislature was one of the first legislative bodies in the United States to reject Federal aid in this field.

CORRESPONDENCE

COMMENT ON DR. O'HARA'S LETTER PUBLISHED NOVEMBER 27, 1930

Editor the *New England Journal of Medicine*,

Will you lend me space for a short medical homily. The text is furnished by a letter from Dr. O'Hara in the *Journal* of November 27, 1930, p. 1104.

What I aim to do is to draw some clinical conclusions where Dr. O'Hara, in his letter at least, appears to be intent on the bacteriology. Thus he seems to incline to the belief that repeated vaccine inoculation influenced the result of recovery in the one case of typhoid as against death in two others where inoculation had not been made. It would be interesting if Dr. O'Hara had let it be known what treatment was given in the two fatal cases. Anything is possible in these days where in a large hospital five chemicals, three of them intravenously, are ordered for a patient who is to be defunct in three days.

The question arises: Is the diagnosis in these milk-borne typhoid epidemics established by autopsy? Dr. O'Hara mentions that two of the cases included had no symptoms. Yet the bacillus typhosus was identified in the blood, hence the diagnosis. I suspect that this is the same epidemic reported by the Massachusetts Department of Public Health— of about fifteen cases, several of which had no symptoms, but merely exposure. Yet in all of these cases the bacillus was caught in the blood. It used to be stated that it was difficult to demonstrate the B. typhos. in the blood. Have methods improved or have we the right to doubt the laboratory? The implicit faith, placed in laboratory and x-ray as a basis of action by the profession, seems to amount to credulity. It requires a stretch of the imagination to think of the blood flushed with a school of spawning bacteria, and no disturbance of the individual!

On finishing a six years' residence in the B. C. H., I wrote a paper on the Spleen in Typhoid Fever—an important factor in diagnosis. It was based on over 1000 cases. Fifteen years later we were talking about the comparative rarity of typhoid—and that was of course before the advent of B. typhos. and vaccines. We did not have any epidemics of typhoid in those days. It was a summer to autumn disease; cold weather found next to no cases in the hospital. Does no suspicion attach to the diagnosis of a sudden tornado of an epidemic, affecting only a certain group of persons?

Dr. O'Hara's case of scarlatina likewise raises some questions as to modernism in medicine. The child had scarlatina, the mother developed facial erysipelas. The doctor expresses chief interest in the (identically same) bacillus producing (?) two different broods. I hope that other reflections are in the background.

Will he use serum in his next case of scarlatina? He had a most severe reaction culminating in a gluteal abscess.

Query 1: Why intramuscular injection of a foreign (thus irritating) fluid? I see nurses giving an ordinary hypodermic in the deltoid. The subcutaneous connective tissue is the safe place in which to deposit the contents of any syringe. The places where the skin is loosened have the choice. Thus over scapula, under the pectoral and the back of the forearm. The latter is used for an ordinary hypo., because, under the loose skin the injected fluid can spread out and this process be assisted, as it ought to be, by pressure and massage.

Query 2: Why, after what seems to have been signs enough of pus, was five days allowed to elapse by the surgeon called in, and a pint of pus to collect, before incision, in these days of "Traumatic" Surgery? (Title of a published surgery.)

Query 3: Why any serum at all? Seeing all the eruptive diseases of Boston, that came to the hospital, in four years, I liked to hear Professor Morse saying, as I have so many times, no such diphtheria today. One can scarcely realize the frightful cases of those days. (Dr. Morse and I were about contemporaries.) With this in mind and with the inclusion of bacteriological cases (and all our cases must surely have a "diphtheria" membrane), how much logic is there in comparative figures of mortality? Or why is a mortality of 8+% much of a showing for antitoxin, a supposed specific antidote, or a 25% mortal-

ity in Minneapolis within a few years? This is worth much more discussion than can be given it.

But equally so with scarlatina.—In point of virulence it is nothing the same. The poison has been attenuated. Just as the scarlatinal mortality of England—as shown by the Registrar's reports (for at least a century I think) has been steadily decreasing.

In Dr. O'Hara's case we have a child with temperature of only 102°—this looks like a very moderate case, scarlatina producing such extreme elevations.

Now what of the virtues of an expectant treatment and wet packs, oiling, warmth, with supporting therapeutics with perhaps a child well in five or six days!

What has become of analeptic and expectant treatment? Of the "natural history" of disease? Of such apothegms as "Vis medicatrix naturae" "Natura duce" (Massachusetts Medical Society's Motto) "Medicus minister, non magister, naturae." (The physician, the underling, not the overlord of Nature.) "Expellas naturam furca, tamen usque recurrit." (You may pitch Nature out yet she always comes back.) Dr. Christian sounds a valiant note of leadership in his annual oration on Skepticism: but I see little notice taken of it. What! Doubt the doctrine of "focal infection"! — "damnable practice of pulling teeth". Why not doubt also antigens and the exquisite chemistry of acidosis as applied to the human?

I inherited a case with this diagnosis (acidosis), that had a vast amount of lung fibrosis and a heart twice normal, to which no attention was called by three specialists. In the epigastrium were two puckered scars of glucose-injection-abscesses. The vaccines of the allergists in asthma, I hear spoken of by patients and their friends, as "knocking them out". The Bacteriology classification of Pneumonia, which has penetrated to the public, seems to lead nowhere. The effort to prove that a serum-treated series shows something better than others, is clearly overstrained when the mortality mounts toward 40%. Pneumonia remains old-fashioned lobar pneumonia, with varying symptom-complexes of different prognostic import.

The Latin mottoes quoted are not catch-phrases. They epitomize the fact that, with nature the original chemist and a myriad of secrets up her sleeve, and with science begging the fundamental question of vitality, there is a wide field for the empirical in medicine, and it is this that makes the physician of hard sense and acquired instinct. The modern practice of medicine is like football, which is a good game but neither a science nor a fine art. To make it seem greater, we have introduced plenty of paraphernalia but where is the bride (pherne)? Just as we should like to see the college bold enough to scrap the paraphernalia of football and return to the simple requirements of a game, so we should like to find the clinician with sufficient determination to run a series of pneumonia, scarlatina and diphtheria on the basis of the simple empirical and analeptic. It is the function of the physician to simplify, not to amplify. I am reminded of a passage from a Latin poet of which I made a version for the *Journal* a long while ago. It concerns one Dr. Symmachus and his students.

I felt a little ill—from fever quite free!
Thou camst, my Symmachus, in the wink of an 'ee.
By an hundred raw pupils thy posse was made
By an hundred hands handled (for the sake of the trade)
An hundred hands touched me; I fetched me a squeal, Oh!
They must have been touched by the frosts of Aquilo.
By my faith, where of fever, there erstwhile was none
Now, Symmachus, I feel it in every damned bone.

Medicine can be "traumatic" as well as surgery (in the title words of the naïve author).

SAMUEL DELANO, M.D.

New Britain, Conn.
January 14, 1931.

THE USE OF DR. OR M.D. BY AN OPTOMETRIST

January 16, 1931.

Editor of *New England Journal of Medicine:*

Referring to the discussion Oculists and Optometrists, page 136, issue of January 15, the following statement is offered.

Correspondence with the Massachusetts Board of Registration in Medicine within two years developed the following reply.

An optometrist may use M.D. after his name if the word optometrist appears beneath, i.e.,

John Doe, M.D.
Optometrist

(or, I think, the title Dr. before the name?).
This is often done and leads to confusion in the public mind. It was not stated whether this was a court decision or a ruling of the board.

Respectfully yours,

W. H. MERRILL.

EDITORIAL NOTE:

There can be no question but that a graduate in medicine who has acquired the degree of M.D. can use that designation in association with his name in connection with the practice of any profession. It is our contention that an optometrist who has not acquired an M.D. degree cannot lawfully use it. We have been informed by telephone with the Executive Secretary of the Board of Registration in Medicine that a letter was sent to Dr. Merrill about two years ago relating to the use of the title Dr. (not M.D.) by an optometrist. A member of the Staff of the Attorney General ruled that an optometrist could use the title Dr. because Dr. or doctor has been more or less generally used by persons engaged in various professional activities. We are informed that the State Board of Optometry discourages the use of the title of doctor by optometrists, but this does not prevent its use. The specific provision of law relating to publicity by an optometrist is found in Section 70 of Chapter 112 General Laws as quoted in a pamphlet put out by the Board of Registration in optometry which reads as follows:

Section 70. Every person to whom a certificate of registration has been granted shall cause the same to be recorded in the office of the clerk of the town where he principally carries on the practice of optometry; and if he removes his principal office from one town to another in the commonwealth, he shall, before engaging in practice in such other town, notify the board in writing of the place where he is to engage in practice, and obtain from the clerk of the town where his certificate is recorded a certified copy thereof and file the same with the clerk of such other town. The fee for recording such certificate or certified copy thereof shall be fifty cents. Every registered optometrist shall display his certificate of registration in a conspicuous place in the principal office wherein he practices optometry, and shall, whenever so required, exhibit it to said board or its authorized representative; and whenever practicing optometry outside of or away from his principal office or place of business, he shall deliver to each customer or person fitted with glasses by him a memorandum of purchase, containing his signature, home post office address and the number of his certificate of registration, together with a specification of the lenses and frames or mountings furnished and the price charged therefor.

The facts seem to be that an optometrist who complies with this section may call himself Dr. but not use the letters M.D. unless he has lawfully acquired that degree.

A REFERENCE TO MEDICINE AND STATESMANSHIP

Editor, *New England Journal of Medicine:*

Apropos of Dr. Haigh's letter and the editorial of January 22, the article "Medicine and Statesmanship" by Lord Dawson of Penn, in the current *Atlantic Monthly* is timely reading.

JOSEPH A. COGAN, M.D.

Warren Chambers,
Boston, Mass.

RECENT DEATH

WARNER—DR. CAMILLUS TURTEN WARNER, a councilor of the Massachusetts Medical Society from 1914 to 1918, died at the Marlborough Hospital following a bladder operation, January 18, 1931, aged 66.

A native of Landgrove, Vt., he was graduated from the Vermont Academy and from the University of Vermont School of Medicine in 1885. Latterly he had practiced Eye, Ear, Nose and Throat work.

He had been a member of the Marlborough Hospital Staff for 21 years and superintendent of the Sunday School of the Baptist Church for 25 years, also he had been a director of the Savings Bank and of the First National Bank of Marlborough. Dr. Warner was a member of United Brethren Lodge of Masons, Mizpah Chapter Order of the Eastern Star, Marlborough Lodge of Odd Fellows, Sons of Union Veterans and of the Marlborough Medical Society.

He is survived by a son and a daughter.

OBITUARIES

GEORGE PIERCE TWITCHELL, M.D.

On December 2, 1930, Dr. George Pierce Twitchell died at his home in Greenfield, following a long period of ill health. In his death the medical profession of Massachusetts lost one of its most capable and respected members, the town of Greenfield one of its most public-spirited citizens, and the Massachusetts Medical Society one of its most honored and loyal workers.

Born in Keene, N. H., in 1855, he was graduated from Harvard College in 1877, and from the Harvard Medical School in 1880. For a time following his graduation he practiced medicine in Boston, but in 1886, he took up his residence in Greenfield, where he continued to practice until his retirement on account of ill health in 1928. In the 44 years of his residence in Greenfield, he had been identified with most of the important medical and health activities of the town. The District Medical Society, the Board of Health, the Franklin County Public Hospital, the District Nursing Association and the Franklin County Public Health Association, all bear witness in one way or another to the many activities of this man. Soon after his arrival in Greenfield he was instrumental in the formation of an active health department for the town, and as one of the original members of the newly elected health board, he helped to establish many measures of great importance to the health and welfare of the town. Although serving as a member of this board for only a limited time, his interest in health matters and disease control never ceased. He was one of the first members of the Franklin County Public Health Association, and was a Vice-President of this organization at the time of his death.

Always greatly interested in hospitals and hospital benefits to the community, he was one of those who contributed largely both of time and energy, in the establishment of the Franklin County Public Hospital at Greenfield in 1895. When new buildings to meet the growth of the institution became necessary, he was appointed chairman of the soliciting committee, and it was largely through his persistent efforts in this direction that these new buildings, coming as a result of his solicitations, were occupied in 1910, with their cost completely paid for. For twenty years he was a member of the Board of Trustees of the hospital and his opinions and judgment were always most excellent, progressive and helpful. As a member of the Massachusetts Medical Society, he had held numerous offices in his County Society, and for the past 25 years, up to the time of his death, he had represented his district in the Council of the Massachusetts Medical Society. As a councilor he was noted for his constant attendance, and it was seldom that the Franklin District Society was not represented by him at the meetings. He had held at dif-

ferent times, various offices in his County Society, including that of President, and he had also been a Vice-President of the Massachusetts Medical Society.

Appointed as Associate Medical Examiner, when the law creating this office went into effect, he succeeded to the office of Medical Examiner in 1899, serving continuously from then until 1922, when he resigned on account of failing health. Combining in a high degree, those qualities of quick perception, keen insight, good judgment and quick decision, qualities so often required in this position, his work in this office had been most exemplary and he was looked upon as one of the best Medical Examiners in the State. He had been a most enthusiastic and active member of the Massachusetts Medico-Legal Society and had served for two years as its President.

In addition to his many activities in a professional way, he had been active socially, taking part in many undertakings carried out by citizens, club and church life. As a member of the Unitarian Church, he had always been keenly interested in all that pertained to the church and its activities, and was one of the most active members of the local Unitarian Layman's League, and had served as one of its early Presidents.

Coming from a family of physicians, all of whom possessed high ideals and were active and progressive in all in which they became interested, it can truthfully be said, that Dr. Twitchell bore out to a high degree, the family traditions, and throughout his professional life, demonstrated in the most excellent way, all that the ideal physician should be, in his relations to his profession, to his patients and to the public.

RESOLUTIONS ADOPTED AT THE JANUARY MEETING OF THE STAFF OF ST. ELIZABETH'S HOSPITAL ON THE DEATH OF DR PIERCE J. DUNPHY

Whereas,—in His infinite wisdom, Almighty God has taken from our midst, in the prime of his active life, our colleague, Dr. Pierce J. Dunphy, whose professional skill and enthusiasm, and lovable personal qualities endeared him to his associates,

It is resolved that—The Staff of Saint Elizabeth's Hospital in meeting assembled, express its sorrow at the loss of a dearly beloved and loyal staff member.

It is further resolved that a copy of this resolution be spread on the records of the Staff, and that a copy be sent to the family of Doctor Dunphy, and to the *New England Journal of Medicine.*

E. J. Cronin, M.D., *Secretary.*

January 8, 1931.

A TRIBUTE TO DOCTOR LUTHER COLBY ROOD

The Boston City Hospital,
January 15, 1931.

Doctor Luther Colby Rood, of the Ophthalmic Staff of the Boston City Hospital, died June 27, 1930, at the age of fifty-two. He was for many years a valued member of our Staff. He graduated from Harvard Medical School in 1899 and spent the following four years in Vienna doing postgraduate work, the most of which time was spent in the clinic of Dr. Ernest Fuchs, an international authority on eye diseases. He then returned to Boston and began practice in ophthalmology.

Besides serving as Ophthalmic Surgeon at the Boston City Hospital, he held the same rank at the Beth Israel Hospital and previously had been an Assistant at the Massachusetts Charitable Eye and Ear Infirmary. He was a member of the American College of Surgeons, the New England Ophthalmological Society and the American Medical Association. He published several illuminating articles on Ophthalmic Surgery.

His service to the Hospital over a long period of years was unselfish and very satisfactory. He was held in high esteem as a consultant by the other services. In his passing away the Hospital loses the services of a conscientious and valuable member of its staff.

P. F. Butler, *Senior Staff.*

NEWS ITEM

HARVARD MEDICAL SCHOOL — At a meeting of the President and Fellows of Harvard College on November 24, 1930, Dr. Frederick Carpenter Irving was elected Professor of Obstetrics at Harvard Medical School. Consent was given by the Board of Directors at a meeting January 12, 1931. This change is to take effect September 1, 1931.

Appointments voted by the Corporation are as follows:

From November 1, 1930 to September 1, 1931:

Richard Hayes Meagher, M.D., Assistant in Surgery.

From December 1, 1930 to September 1, 1931:

Harlan Fay Newton, M.D., Assistant and Austin Teaching Fellow in Surgery.

From January 1, 1931 to September 1, 1931:

Carl Joseph Klemme, M.S., Teaching Fellow in Biological Chemistry.

Richard Upjohn Light, M.D., Arthur Tracy Cabot Fellow in Charge of the Laboratory of Surgical Research.

Resignations to take effect January 1, 1931:

Nathaniel Robert Mason, M.D., as Instructor in Gynaecology and in Obstetrics.

Samuel Bernard Nadler, M.A., as Teaching Fellow in Biological Chemistry.

NOTICES

RADIO MESSAGES

Courtesy WBZA

SPONSORED BY THE STATE DEPARTMENT OF PUBLIC HEALTH

Every Wednesday at 5.20 P. M.

Program ending February 25, 1931.

February 4—"Infantile Paralysis", Dr. William Aycock.

February 11—"Heart Disease", Dr. Richard Cabot.
February 18—"Age and Cancer", Dr. Shields Warren.
February 25—"The Laboratory in Medicine and Surgery", Dr. William A. Hinton.

The Department of Public Health will tell the radio audience about its various activities every Friday—12:30 to 12:40 P. M.

"Health Forum"

Directed by the State Department of Public Health. Courtesy WEEI every Friday at 4:50 P. M. Queries and Answers will be broadcast.

Questions on health and prevention of disease may be sent to:—"Forum", State Department of Public Health, State House, Boston, Mass.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P. M., on Thursday, February 5, in the Amphitheatre of the Peter Bent Brigham Hospital, a medical clinic will be given by Dr. Henry A. Christian, to which physicians are cordially invited. These clinics will be held regularly on Thursday afternoons.

NOTICE OF EXAMINATION FOR ENTRANCE INTO THE REGULAR CORPS OF THE UNITED STATES PUBLIC HEALTH SERVICE

Examination of candidates for commission as Assistant Surgeon in the Regular Corps of the U. S. Public Health Service will be held at Washington, D. C., on March 9, 1931.

Candidates must be twenty-three years and not over thirty-two years of age. They must have been graduated in medicine at a reputable medical college, and have had one year's hospital experience or two years' professional practice. They must satisfactorily pass oral, written, and clinical tests before a board of medical officers, and undergo a thorough physical examination.

Successful candidates will be recommended for appointment by the President, with the advice and consent of the Senate.

Request for information or permission to take this examination should be addressed to the Surgeon General, U. S. Public Health Service, Washington, D. C.

H. S. CUMMING, *Surgeon General.*

CORRECTION

The spelling of Nikola Tesla appeared incorrectly on page 115, issue of January 15, in Dr. Brown's discussion of Dr. Clark's paper.

The above spelling is correct.

REPORTS AND NOTICES OF MEETINGS

WILLIAM HARVEY SOCIETY LECTURE

On Friday evening, January 9, 1931, Dr. George H. Bigelow, Health Commissioner for the Commonwealth of Massachusetts, addressed a large, enthusiastic audience at the Beth Israel Hospital Auditorium on the Subject, "Adult Life." Dr. Clarence L. Scamman was chairman of the evening.

Dr. Bigelow's talk was extremely entertaining due to his many humorous stories and his characteristic method of delivery. The appreciative audience was in a continuous state of smiles, chuckles, and outbursts, as Dr. Bigelow advanced with his address.

"It's a silly subject,—and don't expect much from a health officer," said Dr. Bigelow. He then raised the question as to what an adult really is from the mental, physical, legal and physiological viewpoint. Regarding the last mentioned, he said, "No bells ring, or whistles blow when puberty arrives—it is a vague transformation varying in each individual."

Regarding statistics relating to adult life, communicable diseases caused 16 per cent. of deaths. In death statistics—heart disease was first, followed by cancer and pneumonia. In incidence, rheumatism led heart disease and cancer. The average duration of these conditions was as follows,—cancer 3 years, heart disease 6 years, rheumatic conditions 12-15 years. The economic problems relating to these sicknesses and of what can be done for them were discussed.

The question of how to increase the span of life was raised by Dr. Bigelow. He considered the following,—prenatal care; child and adult hygiene. Under the last he discussed (1) the habits of fat people, thin people and former athletes, (2) the art of eating, (3) rest, (4) smoking, (5) posture, (6) women's fashions and (7) care of feet.

Dr. Bigelow then emphasized the periodic physical examinations and the importance of the modern medical man's position in the problem of adult life.

The next meeting of the William Harvey Society will take place on February 13, 1931, at the Beth Israel Auditorium at 8:00 P. M. The speaker of the evening will be Dr. Frank H. Lahey of the Lahey Clinic, whose subject will be "Goitre." Dr. Edward M. Hodgkins, assistant professor of Surgery at Tufts Medical School, will be the chairman of the evening.

PLYMOUTH DISTRICT MEDICAL SOCIETY

The January meeting of the Plymouth District Medical Society was held at the Brockton Hospital, on January 15, 1931. Superintendent, Frederick M. Hollister, with a committee consisting of Dr. George A. Buckley, Dr. Charles D. McCann, and Dr. Henry J. Lupien received the members of the Society and gave them a very excellent program. There were operations in the surgical room performed by Dr. George A. Buckley, and in the nose and throat department by Dr. John A. Noyes. These operations lasted from nine to eleven o'clock. At eleven o'clock there was a short business meeting. Dr. Frederick W. Murdock presided, giving a short address of

welcome. Dr. Loring B. Packard acted as secretary pro tem. The treasurer's report was read and accepted. Dr. Elmer J. Beaulieu read a splendid eulogy on the late Dr. C. E. Lovell. Dr. Alfred M. Lemay read a very beautiful and touching eulogy on the late Dr. Aurele Roy. Dr. Frederick W. Murdock, president of the society, then called upon Dr. Thomas H. McCarthy, vice-president of the Massachusetts Medical Society, who is chairman of the district legislative committee.

Dr. Thomas H. McCarthy reported that the next meeting would be held on February 5, 1931, at the Commercial Club. He said that he had received letters from Dr. Robert Greenough, president of the Massachusetts Medical Society, former president, Dr. John Bartol and executive assistant to the president, Dr. Thomas O'Brien, saying that they would be present at this meeting and would make short addresses. Dr. McCarthy further reported that another prominent speaker would be present. All of the Senators and Representatives of the Plymouth District will be invited to attend. Dr. McCarthy stated that every meeting so far had been very well attended by the Senators and Representatives of this district and at each meeting they had said that they had received much information from the doctors who are familiar with the bills to be considered. They further said that, after hearing the leading members of the Massachusetts Medical Society explain the bills that were to come before them, they could vote intelligently and that they appreciated the courtesy implied in the invitation. Dr. McCarthy stated that the Plymouth District was the pioneer in this special activity and that the Council of the Massachusetts Medical Society had become greatly enthused over meetings of this kind and hoped that every district throughout Massachusetts would adopt the Plymouth method of educating the Senators and Representatives in regard to bills of an important public health nature that would come before the General Court. This is the only way in which our Society can get any first-class influential support. Dr. McCarthy stated that not only the legislative committee of the Plymouth District would be present but he hoped to have a very large meeting so that everyone would know what was going on. He said that the two principal bills that were to come up this year were the Contraception bill and the Vaccination bill of Dr. Woodward.

Dr. Murdock then threw the meeting open for the reading of case histories of the different members that had come up since the last meeting. He then adjourned the meeting in order that the members might go to the other part of the hospital to see clinical demonstrations. The first case that was brought before the members was a case presented by Dr. John J. Condrick. This was a child five years old that had been in an automobile accident. He had had a fracture of the base of the skull followed by meningeal symptoms. Dr. Condrick gave the child streptococcus serum and eighteen lumbar punctures were performed to relieve the symptoms. The child made a good recovery with the exception of an optic neuritis that still persists. The case was discussed by Dr. Noyes, Dr. Murdock, and Dr. Callahan.

Dr. Charles D. McCann then brought forward three cases of Pyloric Spasm in very young infants. Dr. McCann operated on these children directly after birth. The youngest was five days old and the oldest was eighteen days. All of these cases were diagnosed by Dr. McCann and confirmed by x-ray pictures taken by Dr. Loring B. Packard. In discussing Dr. McCann's cases, Dr. George A. Buckley said these were cases of congenital stenosis of the pylorus. Dr. David Pope demonstrated a case of Carcinoma of the Pancreas. Dr. Frederick L. Weiner demonstrated a case of Pyelonephritis that was secondary to a stone in the ureter. Dr. Henry J. Lupien demonstrated a case of infectious myelitis of a man forty-seven years old. Dr. Lupien stated that after the man had entered the hospital he lost the use of his legs within two days and had not been able to use them since. He said that the prognosis was bad. Dr. Anthony Champ demonstrated the electrocardiogram on a patient. Dr. Loring B. Packard, in closing, gave a talk and demonstration on numerous x-ray plates that he had taken. One was a very excellent showing of arteriosclerosis. Another was one of aortic infarct. He also showed one of an esophageal obstruction, and the last case was one of fracture of the hip. This woman was brought in to illustrate the condition. She was seventy-five years old and the fracture extended from the upper angle of the great trochanter to the lowermost portion of the lesser trochanter.

Dr. George A. Buckley then closed the meeting, having presided over the clinical demonstrations in the absence of Dr. M. Frank Barrett. Dr. Buckley gave a very touching tribute to Dr. Barrett in his devotion to his boy who had just received a very serious injury by being struck in the eye with a hockey stick. Dr. Buckley stated that no one knew at the present time whether the boy would lose his eye but everyone hoped that he would make a good recovery. Dr. Buckley spoke very tenderly of Dr. Barrett's sorrow and said that everyone hoped for the best.

The meeting then adjourned to the Brockton Hospital Dining Hall, where a very excellent banquet was served, free of charge, to all who were present. Between fifty and sixty physicians from all over Plymouth County attended this meeting and great enthusiasm prevailed.

CHARLES G. MILES, *Reporter.*

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The regular quarterly meeting was held at 82 Mechanic street, Fitchburg, Tuesday, January 27, 1931, at 4:30 P. M. for the transaction of routine business only.

"Legislative Night" voted at the July meeting, will be held at the Hotel Raymond, Fitchburg, Friday, January 30, 1931, at 7:30 P. M.

The Senator and Representatives of Worcester North District have been invited as guests.

Dr. Robert B. Greenough, President of the Massachusetts Medical Society, and Dr. T. J. O'Brien of the Committee on State and National Legislation will also be guests.

This is an opportunity for every member of this society to become better acquainted with the Representatives in the Legislature from this District.

R. C. JONES, *President.*
C. H. JENNINGS, *Secretary.*

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital at 8:15 o'clock Tuesday evening, January 13, the amphitheatre being filled to capacity. The meeting was conducted by Dr. James P. O'Hare.

The surgical case was presented by Dr. Mahoney: a fifty-three year old woman with a history of attacks once a year for six years of epigastric pain, lasting one to two days, requiring morphia, unaccompanied by nausea, vomiting or jaundice and no genito-urinary symptoms. Ten days before entry she had a severe attack of pain radiating to the right upper quadrant, scapula, and back, with nausea and vomiting, but no jaundice. The pain lasted three days, the vomiting a week.

Intravenous cholecystography showed stones in the gall bladder with impaired function. Icteric index was 15. Physical examination showed no evidence of cardiac damage. Urine showed no albumen or sediment but specific gravity was low and phenolsulphonphthalein excretion was only 12 per cent. The two hour renal test showed a swing of 1006 to 1010 in specific gravity. Blood urea nitrogen was 40. Shortly after medical transfer the phenolsulphonphthalein rose to 40 and the blood urea nitrogen dropped to 22. A cholecystectomy was done under high spinal anesthesia without incident.

The first two medical cases were presented by Dr. Marks. Case 1 was a 44 year old man whose present entry is for peptic ulcer. He was presented for his past history. Fifteen years ago he was immersed in ice water for twenty minutes and subsequently slept in wet clothes. Three days later his feet and ankles were swollen, then his entire body. He was treated with applications of tincture of iodine and hot packs, and a low fluid intake; a diagnosis of acute nephritis being made. He has been perfectly well ever since and his kidney function is normal as judged by laboratory methods.

Case 2 was a boy of 13 admitted for the second time with swelling of the feet, ankles and face. Past history was questionable for scarlet fever, and positive for tonsillitis. A year and a half ago he caught cold after a swim, and noticed gradual swelling of ankles, feet and face. Because of this, though feeling well, he was sent from school and put on a low protein, salt-free diet. Improvement was interrupted by acute retention. A decapsulation of one kidney at the Carney Hospital was followed by return to apparent health until eight months ago when edema reappeared following a cold and sore throat.

Physical examination showed only edema, pallor and ascites, palpable spleen, and slightly elevated blood pressure. In the urine were a large trace of albumin, numerous hyaline, granular and cellular casts and red blood cells, which later disappeared. Blood urea nitrogen was 7-10 per cent. phenolsulphonphthalein excretion over 40, protein in blood plasma decreased to 3 to 4 grams (albumin being greater than globulin), cholesterol increased to 500 to 900 milligrams from a normal of about 200.

Treatment by the Osman régime was accompanied by gradual improvement and the patient was discharged after three months. One month ago, following a cold and sore throat he was admitted with similar findings.

The third medical case was presented by Dr. Doty of a boy of 18 with a history of frequent head colds the last being four weeks before entry. One week later he had swelling of the eyes and ankles, nausea and vomiting. Examination revealed a dry, pale, pasty skin, moderate edema, slight ascites, some cardiac enlargement and elevation of blood pressure, and hypoactive reflexes. He was placed on a diet containing 40 grams protein and 1800 cc. fluids. Laboratory findings were hemoglobin 40 per cent., red cells 3,400,000, white cells 14,400, slight achromia and poikilocytosis. Urine was acid, specific gravity 1032, large trace of albumin, sediment containing many red and white cells and casts. Blood urea-nitrogen was 32, phenolsulphonphthalein excretion 22 per cent., cholesterol 453, total plasma protein 4.5, albumin 2.4, globulin 2.9. Blood pressure was 158 systolic.

Dr. Henry A. Christian was next introduced as the speaker of the evening. His subject was "Clinical Classification of Nephritis."

The same classification will not suit both the adult and childhood types of nephritis. There are two desiderata for a classification: 1. Reasonable subdivision into groups. 2. A form of subdivision easily applicable to patients, neither too general, nor too definitive. One of the difficulties in classification has risen from a misconception of the etymology of nephritis which does not necessarily refer to inflammation, but is correctly applicable to any disease of the kidneys, regardless of its nature or chronicity. (See Transactions of London Pathological Society, 1889.)

There is little doubt that the first case had nephritis, an acute renal disturbance with marked edema, but recovery with no present evidence of dysfunction. The second case had the same general type of onset but has never entirely cleared up, there being definite evidence of an active process still existing that is subacute. The third case had a recent acute onset, hematuria, anemia, increased blood pressure, change in the plasma proteins with a downward shift of albumin. He will not get well, but will have a recurring, persistent edema. This prognosis is given because cases, which in the acute attack show similar changes in the plasma proteins, nearly always fail to clear up. If the plasma protein falls below 5.2, or the albumin

below 2.3, edema is likely to occur. Other factors may influence this, but those are the critical levels.

A classification that has proven applicable clinically and correlatable pathologically is

Acute Nephritis
Subacute Nephritis
a. With renal edema; nephrosis.
b. Hemorrhagic (hematuria) type.
Chronic Nephritis
a. With renal edema.
b. Without renal edema.
Vascular hypertension progressing into chronic nephritis.

By this a time relationship is provided. Acute nephritis may get well or run into the subacute stage; when with edema it may be called nephrosis provided it satisfies the criteria of this diagnosis: albuminuria, reduction of the plasma protein shift of the albumin globulin ratio, high blood cholesterol, no nitrogen retention, low metabolism. Bright recognized all these peculiarities except the low B M R and the reduction of the albumen globulin ratio.

From Chronic Nephritis without renal edema down, nothing is known about the etiology. The remainder is practically all caused by some form of streptococcus. Pathologically, down to and including chronic nephritis with renal edema, they present some form of glomerulonephritis except for some pure nephroses, rare except in children, which show largely a degeneration of tubules with no or extremely slight glomerular changes. These have glomerular disturbances of a physico-chemical rather than a pathologico-histological type and hence the large excretia of albumin in the urine.

Experiments on the toad-fish, whose kidney has no glomerulus, indicate that, without glomerular injury, albuminuria is impossible.

Renal edema is specified in the classification in contrast to circulatory. The complex mechanism of edema can be understood by stressing certain variants: 1. Filtration pressure. 2. Osmotic pressure. 3. Permeability of the vessel wall. 4. Electrolytes in blood vessels and tissues (practically sodium chloride concentration), and 5. Nervous control, to which great and small rôles are ascribed.

The average normal osmotic pressure of the plasma proteins is 23 millimeters of water. The filtration pressure at the arterial end of a capillary is slightly above 23, at the venous end, slightly below. The normal result is that the outflow into the tissues at the arterial end is compensated by the inflow at the venous end. If the osmotic pressure is reduced, the inflow will be less and the outflow greater and edema will result. If the filtration pressure is increased above normal at the venous end, edema will result. (This is cardiac or circulatory edema.)

Experiments have shown that as blood proteins are lost, the globulin is replaced more rapidly than the albumin, thus explaining the change in the ratio; and when the globulin lags the lipoids increase—a homeostatic mechanism to restore normal osmotic relations. This simple explanation of the blood findings in nephrosis obviates its classification as a separate general disease.

The meeting adjourned at 10:00 P. M.

FIFTEENTH ANNUAL CLINICAL SESSION OF THE AMERICAN COLLEGE OF PHYSICIANS

The Fifteenth Annual Clinical Session of the American College of Physicians will convene in Baltimore, Maryland, March 23-27, and in Washington, D. C., March 28, 1931. The organization holds this Session in Baltimore through the cordial invitation of the Johns Hopkins University School of Medicine, the University of Maryland School of Medicine, the Medical and Chirurgical Faculty of the State of Maryland, the Baltimore City Medical Society, and the further coöperative interest manifested by the various Baltimore hospitals and civic societies. Held in important medical centers, these Clinical Sessions constitute, perhaps, the most important postgraduate week in Internal Medicine each year. Those who attend the meeting will find ample in the way of clinical, laboratory, research and historical interest, well to repay them for the time spent in making the journey. Dr. Sydney R. Miller, of Baltimore, President of the American College of Physicians, has prepared the program for the General Scientific Sessions, while Dr. Maurice C. Pincoffs, General Chairman, also of Baltimore, has arranged the program of clinics, demonstrations, entertainment, etc.

As an added feature of the Clinical Session this year, an additional day, March 28, will be spent in Washington, D. C., where a special program of clinics and inspection tours has been arranged under the auspices of the Medical Departments of the U. S. Army, U. S. Navy, U. S. Public Health Service and Georgetown University. Dr. William Gerry Morgan is acting as Chairman of the Washington Committee, and is being assisted by Admiral Charles E. Riggs, Surgeon General of the Navy; General Merritte W. Ireland, Surgeon General of the Army; General Hugh S. Cumming, Surgeon General of the U. S. Public Health Service; Dr. William A. White, Director of the Government Hospital for the Insane; Dr. John A. Foote, Dean of the Medical Department of Georgetown University; Dr. Ales Hrdlicka, Director of the Department of Zoölogy of the National Museum; Dr. Roy Adams, Chief of the Medical Service at Mt. Alto Veterans' Hospital; Dr. W. H. Hough, President of the Medical Society of the District of Columbia; Dr. C. B. Conklin, Secretary of the Medical Society of the District of Columbia; Dr. George W. McCoy, Director of the U. S. Hygienic Laboratory and Colonel Charles R. Reynolds, Commandant of the U. S. Army Field Hospital School of Carlisle Barracks.

The entire program of the Clinical Session is characterized by new subjects, new authors and wide geographic representation. It is significant that the Committees have attempted carefully to avoid the repetition of subjects and authors, which has so often been the case in previous years, not only on the program of the American College of Physicians, but on the program of a great many medical organizations.

On the General Scientific Programs there will be forty-five or fifty selected formal papers. Symposia on blood diseases, oxygen therapy, diseases of the liver, recent advances in endocrinology with particular reference to the newer work on suprarenal extracts, myocarditis, and several other subjects have been arranged. At Baltimore's many modern and excellently operated hospitals, clinics, ward-walks, laboratory demonstrations and the like will be held. Johns Hopkins Hospital and Medical School, under Dr. Alan M. Chesney, Dean, and a specially appointed Committee, will place at the disposal of the College all of its facilities and offer a program of great interest. Additional hospitals, such as the Union Memorial Hospital, St. Agnes Hospital, at which Dr. Joseph C. Bloodgood does so much of his work, the Municipal Hospitals, and several of the more private institutions. such as the Howard A. Kelly Hospital, noted particularly for its radium activities, and the Sheppard and Enoch Pratt Hospital, which is one of the most modern dealing with psychiatric problems, and many others will provide programs of clinics.

Hotel headquarters will be at the Lord Baltimore Hotel, while general headquarters, at which the registration of members, commercial exhibits and all general sessions will be held, will be The Alcazar, Cathedral and Madison Streets, Baltimore. Transportation on the Certificate Plan of reduced fares will be available to all physicians and dependent members of their families from all parts of the United States and Canada. A special program of entertainment has been arranged for visiting ladies. The Convocation for the induction of new members, as Masters or Fellows, will be held on Wednesday evening, March 25, and the Annual Banquet will be held on Thursday evening, March 26. The Business Meeting, at which reports of administration and elections for the new year will take place, will be held during the forenoon of Thursday, March 26.

Mr. E. R. Loveland, 133-135 S. 36th Street, Philadelphia, is the Executive Secretary of the College, and it is to him that requests for further information or programs should be addressed.

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building, on Thursday, February 12, 1931, at 8:15 P. M.

PROGRAM

1. Presentation of Cases.
2. Methods of Estimating the Degree of Sympathetic Vasoconstriction in Peripheral Vascular Diseases. Dr. John J. Morton, Rochester, N. Y.
3. The Effect of Peripheral Nerve Block on the Surface Temperature of the Extremity. Dr. Reginald H. Smithwick.
4. Résumé of the Results of Operations on the Sympathetic Nervous System at the Massachusetts General Hospital. Dr. James C. White.

The Discussion will be opened by Dr. Arthur W. Allen.

Physicians, medical students and nurses are cordially invited to attend.

COMMITTEE ON HOSPITAL MEETINGS.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

The next regular meeting of the Norfolk South District Medical Society will be held at the Norfolk County Hospital, South Braintree, on February 5 at 12 noon.

The speaker will be Dr. Francis B. McCarthy whose subject will be "Focal Infections."

N. P. PILLSBURY, *Secretary.*

NEW ENGLAND ROENTGEN RAY SOCIETY

The February meeting of the New England Roentgen Ray Society will be omitted owing to the joint meeting to be held in Baltimore February 6 and 7.

T. R. HEALY, M.D., *Secretary.*
370 Marlborough St., Boston, Mass.

CARDIAC COURSE

IV. ELECTROCARDIOGRAPHY

1. Methods of Electrocardiography and the Normal Electrocardiogram. Dr. James M. Faulkner, Boston.
2. The Use of Electrocardiography in the Study of Arrhythmias. Dr. E. H. Drake, Portland, Me.
3. The Clinical Significance of Abnormalities of the Electrocardiographic Complexes. Dr. Lewis Hurxthal, Boston.
4. Discussion to be opened by Dr. F. Dennette Adams, Boston.

The fourth meeting of the Cardiac Course of the New England Heart Association will be held on Wednesday afternoon, February 4, at four-thirty o'clock in John Ware Hall at the Boston Medical Library, 8 The Fenway. All physicians and students interested are cordially invited.

GREATER BOSTON MEDICAL SOCIETY

A joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital will be held on Tuesday, February 3, 1931, at 8.15 P. M. at the Beth Israel Hospital.

Program: The Rôle of Obstructive Lesions in the Urinary Infections of Childhood. Dr. Charles Mixter, Chief of Surgical Staff, Beth Israel Hospital.

Discussion: Dr. J. Dellinger Barney, Massachusetts General Hospital; Dr. William Carter Quinby, Peter Bent Brigham Hospital.

BERNARD I. GOLDBERG, M.D., *Secretary.*
483 Beacon Street, Boston.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

January 30—Belmont Medical Society. See notice on page 189, issue of January 22.

February 2—American Heart Association, Inc. See details on page 139, issue of January 15.

February 2-March 30—Lectures on Keeping Mentally Fit. Given under the auspices of the Massachusetts Division of University Extension and the Massachusetts Society for Mental Hygiene. Boston.
Eight lectures, in the Gardner Auditorium, State House, Boston, Monday evenings, beginning February 2, 1931.

Course Leader: Dr. Henry B. Elkind, Medical Director, Massachusetts Society for Mental Hygiene.

February 2—Keeping Mentally Fit: A New Art and Need of the Times. Joseph Jastrow, Ph.D., Formerly Professor of Psychology, University of Wisconsin.

February 9—The Child Is Father to the Man. James S. Plant, M.D., Director, Essex County Juvenile Clinic, Newark, N. J.

February 16—Fatigue, Worry, and "The Blues." Karl M. Bowman, M.D., Assistant Professor of Psychiatry, Harvard Medical School.

March 2—Do We Americans Really Live? James J. Walsh, M.D., Medical Director, Fordham University School of Sociology, and Professor of Physiological Psychology, Cathedral College, New York City.

March 9—The Job and Mental Health. Elton Mayo, Harvard Graduate School of Business Administration.

March 16—Emotion and Intellect in Adult Life. William B. Terhune, M.D., Austen Riggs Foundation, Stockbridge.

March 23—Normal and Abnormal Fear. Abraham Myerson, M.D., Professor of Neurology, Tufts College Medical School.

March 30—"Nerves"; Their Meaning in Our Lives. E. Van Norman Emery, M.D., Medical Director, Connecticut Society for Mental Hygiene.

February 3—Joint Meeting of the Greater Boston Medical Society and the Beth Israel Hospital. For detailed notice see page 241.

February 4—Cardiac Course. See page 241.

February 5—Medical Clinic at the Peter Bent Brigham Hospital. See page 237.

February 11—The New England Dermatological Society. See page 189, issue of January 22.

February 12—Massachusetts General Hospital. See page 241.

February 13—The William Harvey Society at the Beth Israel Hospital. Dr. Frank H. Lahey will speak on "Goitre". See page 237.

March 13—The William Harvey Society at the Beth Israel Hospital. Dr. Samuel R. Meaker will speak on "The Social Aspect of Gynecology".

March 23-27—Fifteenth Annual Clinical Session of the American College of Physicians. See page 240.

May 11-14—Meeting of the National Tuberculosis Association at Syracuse. See page 1310, issue of December 25.

July 27-31—Third International Congress of Radiology, Paris. Complete notice appears on page 385, issue of August 21.

DISTRICT MEDICAL SOCIETIES

Bristol North District Medical Society

April 16—Taunton Woman's Club.

September 17.

Essex South District Medical Society

Wednesday, February 4—Council meeting, Boston.

Wednesday, February 11—Danvers State Hospital. Clinic at 5 P. M. Dinner 7 P. M. Speaker: Dr. Bryant E. Moulton, Assistant Director of the Judge Baker Foundation. Subject: "Juvenile Delinquency."

Wednesday, March 4—Lynn Hospital. Clinic at 5 P. M. Dinner at 7 P. M. Speakers: Dr. Henry H. Faxon, Boston; subject: Treatment of Varicose Veins and Ulcers by the Injection Method. Dr. Arthur W. Allen, Boston; subject: Neurovascular Surgery.

Thursday, May 7—Censors meet at Salem Hospital at 3:30 P. M.

Tuesday, May 12—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. Morris Fishbein, Editor of the A. M. A. Journal of Chicago. Subject: "History of Medical Cults." Ladies invited.

Franklin District Medical Society

March-May—The meetings are held on the second Tuesdays of March and May at 11 A. M. at the Weldon Hotel, Greenfield.

Middlesex East District Medical Society

March 11—At Reading.

May 13—At Wakefield.

Norfolk District Medical Society

February 24—Dr. F. C. Irving and Staff, "Difficult Labor Cases and Indications for Interference." Boston Lying-in Hospital, 8 P. M.

March 31—Dr. Arthur W. Allen. Subject to be announced (will deal with circulatory diseases). Hotel Kenmore, 8 P. M.

May—Annual Meeting. Date and details to be announced.

May 7—Censors' Meeting for examination of candidates at 171 Warren Street, Roxbury, 4 P. M. Candidates for schools on accepted list will forward application with certificate of graduation to the secretary at least one week prior to date of examination. Candidates from schools not on accepted list who have been in practice five years will forward applications together with five letters of recommendation from members in good standing in the Massachusetts Medical Society to Dr. P. P. Johnson, Cabot Street, Beverly, Mass., two weeks prior to date of examination. Fee for all candidates $10.00.

FRANK S. CRUICKSHANK, Secretary.

Norfolk South District Medical Society

February 5—See page 241.

Other meetings for the ensuing year are as follows: March 5, April 2, May 7.

All meetings begin at 12 noon. Meetings will be held at the Norfolk County Hospital, South Braintree.

The censors will meet on May 7 at 11:30 A. M. at the Norfolk County Hospital, South Braintree.

NATHAN R. PILLSBURY, Secretary.

Plymouth District Medical Society

February 5—See page 238.

Suffolk District Medical Society

February 25-April 29—Combined meetings with the Boston Medical Library. These meetings will be held as specified below on Wednesdays, at 8:15 P. M.

February 25—Clinical Program, Boston City Hospital.

March 25—Clinical Program, Beth Israel Hospital.

April 29—Annual Meeting. Election of Officers. Boston Medical Library.

The medical profession is cordially invited to attend all of these meetings.

GEORGE S. DERBY, M.D.,
President, Suffolk District Medical Society.
LELAND S. McKITTRICK, M.D.,
Secretary, Suffolk District Medical Society.
HILBERT F. DAY, M.D.,
Boston Medical Library.

BOOK REVIEW

Methods and Problems of Medical Education. (Eighteenth Series.) The Rockefeller Foundation, New York.

It is impossible to give a brief review of this interesting volume, consisting of thirty articles covering three hundred thirty pages and including many illustrations, plans of buildings and photographs. In the prefatory note to the First Series, attention is called to the increasing difficulties, year by year, of publishing certain types of medical papers. "This is especially true of contributions in the field of medical education, which, in that they are fundamental to all progress, are in the long run of prime importance." It is because of the small number of persons to whom such papers are of direct interest that the publication is difficult, that is to say, expensive.

The Rockefeller Foundation, therefore, publishes year after year, selected papers which are invaluable to students of medical education. In the present series, Europe, Asia, Africa, North and South America, are represented. The sciences are given the larger number of titles, physiology with physiological chemistry and biochemistry, having ten out of thirty. The magnitude of the undertaking is indicated by the thirteen pages of titles of papers that have been published in this way since the inception of the project in 1924. Six sections are devoted to various aspects of the work at the University of Michigan. Perhaps the most interesting to the physician who is not a specialist in medical education are the descriptions of the "Faculty of Medicine," Chulankarana University, Siam, and "La Nouvelle faculté de Médicine" of the University of Brussels, Belgium.